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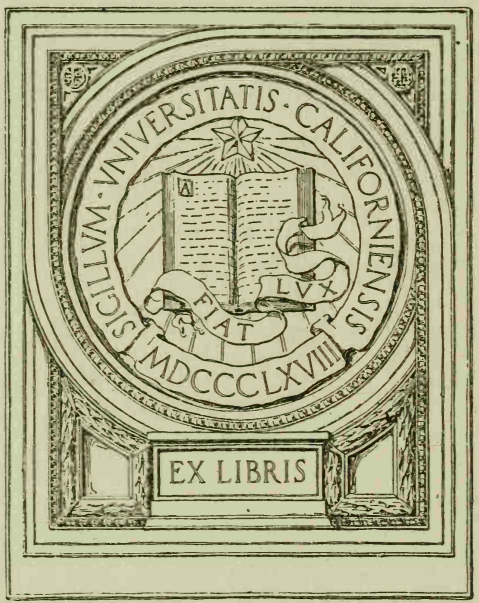

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DISEASES OF THE LUNGS

(OF A SPECIFIC NOT TUBERCULOUS NATURE)

ACUTE BRONCHITES; INFECTIOUS PNEUMONIA; GANGRENE, SYPHILIS, CANCER AND HYDATID OF THE LUNGS

BY

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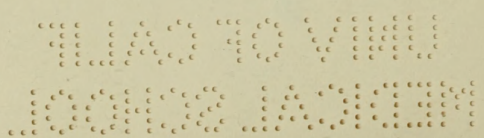

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AUTHOR'S PREFACE.

SINCE the commencement of this century three principal phases have marked the successive stages in the progress of the evolution of scientific medicine. The first, inaugurated by the immortal Laennec, associated the positive discoveries of pathological anatomy with the clinical observation of the patient.

Cruveilhier, Bouillaud, Stokes, Bright (to mention only the most important names), established during this period the bases of modern pathology. In the second phase histology and pathological physiology essayed to explain the morbid types which observation had created, but only succeeded in stating with precision the details of known lesions without being able, except in rare instances, to penetrate the secret of their pathogenetic conditions. The study of causes appertains to the third phase,—to the present epoch.

I have endeavored, as far as lay in my power, to urge onward the movement which to-day draws all progressive spirits toward those fruitful researches from which have already issued so many valuable discoveries.

I have never ceased to teach that histology, abandoned to itself and separated from experimental physiology, must remain impotent and sterile; and that in pathology as in therapeutics, experimentation alone can furnish the notions indispensable for an exact appreciation of the phenomena observed.

Pasteur and Koch, in opening before us the world of microbes and in revealing the all-influential rôle of these minute organisms in the development of diseases, have brought to view a new field for experimentation. The results we are now realizing. We know to-day what is the real cause of septicæmia, of purulent infection, of anthrax, of tuberculosis, of cholera. The words *miasm*, *virus*, under which we have so long concealed our ignorance, are destined to disappear from scientific medicine.

To this vague idea of an impalpable and immaterial cause of the disease

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we have substituted the knowledge of a visible thing able to reproduce and multiply itself ad infinitum, but which may also be attacked and destroyed by appropriate means.

Certain writers have thought that these means were not applicable when the parasite has once grafted itself on the human organism, not being able to reach it in the recesses of the tissues; that consequently the new researches are without any real utility for practical medicine.

Doubtless we do not yet possess the medicament with which directly to antagonize each parasite, and the discovery of the microbiotic cause of the disease does not imply the immediate discovery of the specific remedy which shall destroy it. But is this a sufficient reason for denying the possibility of any such realization in the future? Will any one seriously maintain that the exact knowledge of the etiological conditions of a disease can be of no practical utility, and that while waiting for the discovery of the specific medicament, such knowledge may not serve of advantage in establishing the rules of a rational prophylaxis? Do not the statistics furnished by surgeons and obstetricians give a triumphant response to these too hasty objections? In fine, the discovery of attenuation of virus, for which we are indebted to Pasteur—does it not show us what therapeutics may have a right to expect in the future from such studies, in the way of protection against at least a certain number of infectious diseases?

Many difficulties remain to be met—no one disputes this, but the progress already realized is immense; therefore I have not hesitated to take the data actually gained to science for the basis of my work.

The treatises on pathology thus far published have established their classification and their descriptions according to the nature of the anatomical lesions. It is upon the microbiotic etiology that I have desired to build the foundations of my nosology.

Earnest advocate of the parasitic doctrine I have endeavored to apply the same data to the elucidation of the problem of treatment. This book is necessarily incomplete, since it marks the first phase of a period of transition, but my end will have been attained if it shall have prepared the way for future acquisitions, and if making known what has already been done it shall clearly show what remains to be done.

PARIS, *June 17th*, 1885.

TRANSLATOR'S PREFACE.

On the surface and in the midst of a fluid containing organic matter undergoing decomposition, are seen with a powerful microscope swarms of living parasites, first called by Mulder, monads. Some of these resemble minute granules with Brownian movement, while the greater part look like tiny infusoria and have an active motion of their own. Multitudes of short, staff-like bodies are present, and if the liquid shall have stood some time in a warm room freely exposed to the air little living things of a variety of shapes will have made their appearance. Prof. Cohn, in common with many others, arranges all these living particles under the genus bacterium. Béchamp calls them microzymes. It is not necessary that the putrefying fluid shall be exposed at all to the air. You may tightly cork a fresh organic infusion that to-day contains no bacteria, or even germs, that are to be seen, and in a few days you will have the liquid swarming with living things.

Whence come they, these "infinitely little" organisms? Are they animal or vegetable? Without stopping to review all the phases of the historical evolution of this subject, it is sufficient to state that it is now admitted by all competent authorities that the living particles seen in decomposing fluids are fungus germs, allied to the moulds. They do not arise in these fermenting or putrefying liquids by spontaneous generation. Pasteur's and Tyndall's experiments have conclusively proved this; if you will take pains to sterilize any organic liquid (boiled urine, meat infusion, etc.) it may be kept with entire freedom from decomposition in ordinary temperatures for years. Panspermism is to-day the dominant doctrine; all microbes seen in animal fluids or tissues undergoing retrograde metamorphosis are the product of ova or spores introduced from without. The air is the great receptacle of these germs, which form a part of the dusty column that illuminates every sunbeam. If aught of incredulity should be manifested, as though the relatively few spores discoverable in

the atmosphere were insufficient to accomplish the stupendous results affirmed, we are reminded of the extraordinary fertility of the lowest organisms, whose reproductive activity under favoring conditions is inconceivable.

These spores we may draw in with every breath, we may swallow with every draught, and the higher life is everywhere interpenetrated by the lowest life.

Admitting the cryptogamic origin of these living monads, what is their relation to fermentative and putrefactive changes out of the living organism? What is their relation to the organism in health? What is their relation to the organism when under disease?

I. The term fermentation is generally used in a restricted sense to denote the decomposition of complex (ternary or quaternary) organic substances (milk, sugar, urine, etc.), their descent to a lower chemical plane, with evolution of carbonic acid, but without the evolution of any gases of offensive odor. When organic substances (and especially such as are azotized) disintegrate, their dissolution being attended with evolution of gases of offensive odor, we speak of the change as putrefaction.

Among the fermentations we may specify the alcoholic, acetic, lactic, butyric, gallic, as being among those best known; the connection of each of these fermentations with a definite micro-organism as a causal factor, is a datum now gained to science, and for which we are especially indebted to the labors of Pasteur. It may now be asserted fearless of contradiction that, under all ordinary circumstances, fermentations occurring out of the living body are the result of the vital operations of just such microphytes as we behold in a drop of any decomposing liquid, it being premised that these bacteria are apparently multitudinous in kinds and in functions.

There is a marked resemblance between fermentation and putrefaction in the profusion of living particles developed during those processes; that alcoholic fermentation, for instance, is the result of disturbances set up in saccharine solutions by the *saccharomyces cerevisiæ* has been generally conceded since Cagniard de la Tour first demonstrated that yeast is composed of these torulæ; that the acetic fermentation is dependent on the *mycoderma aceti,* the fermentation of milk on the *lactic vibrio,* etc., are equally susceptible of scientific demonstration. Moreover countless experiments, of which the familiar instance of canned meats is a sufficient example, have proved that the same relation holds between putrefying

substances and the microscopical organisms that are found in them, so that the proposition *without bacteria no putrefaction* expresses the rigorous truth.

II. The question what relation these microscopical beings sustain to higher organisms in health is more easily answered to-day than it was a few years ago. Our knowledge of the "infinitely little," their life history, their habits, their habitats, has wonderfully increased of late, though still very imperfect. Mycology is beset with difficulties which human genius and industry are but slowly surmounting. Whether the ordinary fungi that seem to us so harmless may under certain circumstances develop into the deadly micro-organisms that devastate human society we know not. Certain it is that many species appear to be inoffensive to everything that has life and vigor, while they feed on dead or decaying substances. Some of the moulds,—aspergillus glaucus, mucor mucedo, pencillium glaucum, etc., are of this character. Filiform cells and interwoven filamentous tissue (cells and mycelium) are almost the sole element of these fungi, which are *aerobic,* i.e., can only live in free air. Other fungi are innocuous to animal life while devouring the vegetal. It is known that the whole tribe of rusts and mildews do attack vigorous plants. The *Botrytis infestans* causes the potato rust, and the *Oïdium vitis* is the pest of vine-growers. In fact all vegetables have their peculiar parasites. The same is true of all animals; the highest are a prey to the lowest *where the conditions are favorable* for the nutrition and development of the latter, a state of perfect health being in general unfavorable.

In reality health may be regarded as the expression of the vital integrity of an organism, whose anatomical elements are able successfully to resist the incursions of the lower life. In the individual as in the race, to the higher and more evolved victory is temporarily assured, though defeat in the end is certain.

The immunity which the more complex and more highly evolved organism possesses against the harmful assaults of the living ferments is relative; 1. To the quantity of the assailants. It is evident that in the struggle for existence between the living cells of an organic aggregate and the septic or pernicious microbes, the latter will be much more easily overcome if few in numbers, while like the strongest and best disciplined army the healthy living cells may be out-flanked and overcome by an ever-augmenting foe. In whatever way bacteria may be introduced into the economy, or whatever the kind, it is quite possible for the healthy organ-

ism to tolerate them with impunity provided the quantity be inconsiderable.

2. To the mode of introduction. The fermentiferous, putrefactive or morbiginous bacteria may be inhaled or ingested by a healthy individual without the least harm accruing thereby to the functions of the economy, provided that the fluids and tissues be in a normal state, and *there be no peculiar receptivity.* In fact the ordinary air of respiration contains *septic* germs, and sometimes *disease* germs. The latter, when infesting the atmosphere of inhabited districts, are, as we know, breathed with impunity by multitudes of non-susceptible persons. With regard to the introduction of bacteria into the alimentary canal, it is certain that when the stomach is healthy, the gastric juice is inimical and even destructive to them all. Moulds and septic bacteria may be often eaten without detriment. The Kalmuck Tartars live on raw putrid fish or flesh of carrion, and they are said to be a healthy race; now such putrid meat abounds in bacteria. Koch claims that in perfectly healthy individuals even the cholera microbe when swallowed in food or drink is destroyed in the stomach; it is only when the powers of that viscus are enfeebled and there is deficiency of the natural secretion, that the "comma bacillus" is able to penetrate the intestines and accomplish its mischief. Decroix even dared to eat the meat of an animal that was killed while suffering from splenic fever, and he ate it with impunity.

The injection under the skin or into the blood of bacteria, and especially of such as are of great known virulence, is much more dangerous, but even such inoculation has been practiced with impunity. The ordinary septic microbes when so introduced are prone to give rise to that terrible disease, septicæmia—especially when injected in quantity sufficient to overcome the resistance of the animal cells—but instances are not few where their inoculation has caused but slight (if any) temporary disturbances.

The results of the injection of the morbiginous bacteria may equally be nil; everything depends on the receptivity of the organism. Some persons have a natural or acquired immunity against certain malignant parasites which when introduced into the bodies of other individuals speedily find there a congenial soil. Science is not yet in possession of any very satisfactory explanation of facts of this kind; certain it is that "receptivity" is far from being constant, and that the solids and fluids of the economy are quite soon exhausted of some supposed mineral or organic

principles on which the life of the microphytes depends. Hence the protective value of previous attacks and preventive vaccinations.

III. "He that thoroughly understands the nature of ferments and fermentations," says Robert Boyle, "shall probably be much better able than he that ignores them to give a fair account of divers phenomena of certain diseases (as well fevers as others), which will perhaps never be understood without an insight into the doctrine of fermentations." Although all general diseases are[1] accompanied by qualitative alterations in the blood there are certain diseases in which the blood-changes so markedly partake of the nature of decomposition that it is proper to speak of them as *fermentations;* the analogy between the circulating fluids in these diseases and liquids undergoing fermentative decomposition out of the body being most striking and complete. The question, then, what is the relation between bacteria and the higher organisms when under disease? resolves itself into the subordinate question (the only form in which it can be intelligently discussed)—what is the relation between bacteria and certain diseases known as *Zymotic* (from Ξυμος, *fermentation*)? The term *zymotic* is an appropriate one, for the blood of victims of these maladies (which include the most malignant epidemic and endemic fevers and even tuberculosis and cholera) is invariably found to have undergone to a greater or less extent certain gross changes, such as corpuscular disintegration, decrease of fibrine, increase of carbonic acid, diminution of oxygen; in some of the most malignant of these diseases it becomes tarry, non-coagulable and sticky, and speedily putrefies when withdrawn from the body. There is another characteristic which assimilates the blood of a zymotic affection to a fermentating fluid, i.e., its power of communicating its virus, which may multiply *ad infinitum* in a suitable medium. Take for instance a drop of anthracoid blood and inoculate with it a healthy animal; you may communicate the disease to this animal, which may infect a whole flock, and spread the pestilence over an entire country. Or if this drop of splenic-fever blood be sown in sterilized yeast-water and cultivated and a drop of this culture fluid be sown in another flask of yeast infusion and so on, you will obtain a product which in the fiftieth generation is just as virulent as the original anthracoid blood. This is precisely the case with fermentations taking place out of the body. "A little leaven leaveneth a whole lump." The yeast torula is capable of indefinite

[1] Cited from the Life of Louis Pasteur, New York, 1885.

propagation in a suitable medium. So with the butyric, acetic, lactic ferment. So far, then, the analogy is complete between the viruses which produce measles, small-pox, syphilis, etc., and the living agents of the respective fermentations; they are all capable of unlimited self-propagation. Are there any chemical poisons not of an animated, figured nature, but of a soluble kind, which are known to produce similar results? I think that we may answer this question in the negative, provided it shall not yet appear that the virus of canine rabies and of snake bites are soluble ferments, of the nature of ordinary toxic neurotic agents, but unlike the latter in their ability to multiply themselves in the blood and tissues indefinitely. It would seem from Sir James Fayrer's researches on Snake Poisons (London Lancet, 1st part, 1884) that the blood of a victim of the bite of a venomous snake is infecting, like the original virus. It is known that the virus of rabies is capable of similar diffusion, and is inoculable from animal to animal. While, then, there seems to be every presumption that the active principle of these viruses is a microbe, there is no proof that such is the case. Sir James Fayrer inclines to the opinion that it is a poison, of the nature of *ptomaines;* Pasteur believes an unknown microbe to be the cause. There remains here a lacuna yet to be filled.

But the analogy does not stop here. The alcoholic, lactic and other like fermentations are each due to a particular bacterium, and if the specific fevers are the expression of fermentative changes in the blood, they must each have a different causal microbe. This early prevision of scientific medicine, which has now for the past twenty years been the guiding hypothesis of workers in pathogeny, is now finding justification and verification every day.[1]

It may here be remarked, moreover, that the underlying assumption that the bacteria are of multitudinous species, living under different conditions, thriving in different media, having a different life history, varying in their action on the higher organisms, some being benign, others malignant, some living and multiplying where others perish, is also proved to have scientific validity.

Every one who has intelligently noted the labors of the past twenty years in all lands by indefatigable workers in the domain of mycology and

[1] Any coincidences between certain parts of this Introduction and certain articles which have during the present year appeared in the Boston Medical and Surgical Journal are explained by the fact that I am responsible for those articles.

microscopy, labors conducted with elaborate culture-apparatus and chemical appliances, and costly experimentation, and performed with one great end in view—to ascertain the causes of certain communicable diseases—must have passed through a stage of scepticism to a firm persuasion that progress has been made, and that a clue has been found to many of nature's profoundest mysteries.

Premising that theories and hypotheses are only of use as they are the stepping stones to inductions which demonstrate their truth or falsity, we would here remark that only so far as the practice of medicine is based on correct views of causation can it be anything but empiricism, and if any one thing is clear it is that the drift of scientific therapeutical endeavor must be away from empiricism to treatment founded on known laws—the goal may be distant and difficult of attainment, but the struggle toward it will ever go on.

The analogical argument from which the conclusion is drawn that all the infectious diseases are due to a living ferment, is very strong. In its application to man, this argument presents itself under two aspects: 1, under this syllogistic form—All fermentations owe their efficient causation to microbiotic action; The specific febrile diseases of man are of the nature of fermentations: Therefore they are microbiotic. The other aspect is the following: The epidemic and contagious diseases of plants and the lower animals are, as far as is known, of parasitic origin. The general laws of these epidemics are the same as those which characterize human zymotic diseases.[1] Our belief in the uniformity of nature would lead us to infer that they must have a like causation.

It cannot, however, be too much insisted on that medical truths are not determined by the deliverances of reason, but by inductive demonstration. The specific morbific fungus must be found, and its causal relation to the disease in question proved. Simple concomitance is not enough, for bacteria may attend diseases as birds of prey do armies, to batten on the spoil. There are certain species of microbes that are known to act merely as the scavengers of nature, living on and removing decaying and putrescent matter, and it is legitimate to reason that all microbes have only this function, unless experiment shall prove the contrary, by isolating

[1] The infectious diseases of the lower orders and of man occur sporadically and epidemically. Both are limited to certain conditions of soil and climate, and are especially prevalent during certain seasons of the year. They are dependent on heat and moisture; have a stage of incubation, are self-limited and are, as a rule, non-recurrent.

the suspected micro-organism, separating it as far as possible from all extraneous matters which might possibly have a septic influence, and by inoculations practiced in healthy animals with this microbe, reproducing the disease with which this bacterium was found to be a constant accompaniment. This has been done : 1, in accordance with the method of Chanveau, whereby all the soluble parts of virus are removed by a clay or plaster filter and the solid corpuscles which remain in the filter inoculated, after being washed, in animals; 2. by the method of successive cultures. This is the procedure which is generally resorted to by mycologists. The supposed morbiginous parasite has been found, isolated and cultivated in a proper medium, such as yeast-water, chicken broth, gelatine. An infinitesimal product of one culture has been used to start another culture in a sterilized medium, and so on for many successive generations, and with a minute portion of the final culture, healthy organisms of the same or of different species have been inoculated and the original disease has been reproduced. By such proof Pasteur showed that the epizoötic known as *pebrine*, so prevalent among silk-worms, is the result of the ravages of a microbe discovered by Fillippi, and called by Lebert, *panhistophyton*. Nothing can be more interesting to the naturalist and to the physician than the chapter in the recent Life of Pasteur by his son-in-law, where are detailed the experiments of this savant in the etiology and prevention of pebrine, a disease from which the silk-worm industries of France are now practically free, owing to Pasteur's labors. Another infectious disease, chicken cholera, has been shown to originate in the depredations of a little micrococcus, which is easily cultivated with entire preservation of its virulent properties in chicken broth made alkaline by potassa. It is hardly necessary to refer to the fact that the grape-vine disease (phylloxera), the ergot of grain, the potato disease, have been proved to be caused by microscopic fungi.

The investigations in this country of Salmon and Detmers have conclusively shown that swine plague and the Texas cattle fever are of bacterial origin.[1] Moreover the recent experiments of Schutz and Löefler in Germany, of Charrin and Capitan in France, have demonstrated the bacillary origin of glanders.

Salmon has made many interesting experiments confirmatory of Pasteur's discovery of the microphytic origin of fowl cholera, and justly ob-

[1] Reports Dep. Agriculture, 1880–1883.

serves with reference to the power of propagation of infinitesimal portions of contagion: "Formless ferments are unable to reproduce themselves. Reproduction and multiplication is a function of living matter, and of this alone, and when we have proved that virus can be cultivated indefinitely, it is equivalent to demonstrating that its essential constituent is a living thing."[1]

Among the communicable diseases which prevail among the lower animals is one which is also common to man, though always derived from a diseased animal. I refer to anthrax, also known as splenic fever, charbon, wool-sorter's disease, malignant pustule. This disease, endemic in some parts of Europe, has been observed in this country. Its ravages among cattle and sheep have been very great; in one Russian district alone there perished in the course of three years 56,000 domestic animals and 528 human beings.[2] Severe constitutional symptoms of a typhoid character attend the development of this malady, which may almost be looked upon as the type of acute contagious diseases.

The attempts which have been made these late years by careful, conscientious and indefatigable workers, such as Pasteur, Toussaint, Greenfield, Chanveau, Bert, Klein, Cohn, Duguid, and Burdon Sanderson to ascertain the hidden cause of this disease, have been followed by results as gratifying as they are surprising. Not only has the microbiotic origin of anthrax been proved beyond the possibility of cavil, so that all authorities worthy of any respect admit it, but the specific bacterium, the *bacillus anthracis*, has been found and cultivated, and with the fungous product of a long series of cultures the disease has been reproduced at will. Nay, more, a "protective attenuated virus" has been obtained by suitable cultures in which the modifying effect on the bacillus of exposure to oxygen has been utilized, and by the influence of this preventive vaccine, Pasteur has saved the flocks of Europe by the hundreds of thousands. Moreover the benefit in the way of prophylaxis which has been derived from these enlightened views as to causation, has been incalculable, and even in the case of infectious diseases whose microbe is still unknown, the possibility has, I believe, not seldom been realized, of stamping out the malady by annihilating the cause. A belief in the truth of the "germ theory" of disease underlies the best work of our modern Boards of Health; and the assumption is justified by the results. Listerism in our hospitals applies

[1] Report of the Commissioner of Agriculture, 1881–1882.
[2] Belfield, Cartwright Lectures, 1883.

the germ theory to surgical practice, with brilliant success. Although the specific microbes of surgical and puerperal infection are but partially known, the influence of bacteria of some baneful kind on septicæmic processes is none the less recognized, and the results of strict aseptic treatment, germicide sprays, germicide dressings, and absolute cleanliness are most gratifying. Under Listerian precautions, joints are opened with impunity, resections are performed with boldness, and operations on the pleural or abdominal cavity have ceased to be a terror; ovariotomy is no longer the hazardous thing it once was, cases of septicæmia and purulent infection after operations, of surgical erysipelas and gangrene, are almost unknown in our hospitals.

We must not, however, forget that the acquisitions actually made in the knowledge of the true causes of the specific febrile maladies are still meagre. The inference is indeed irresistible that if anthrax be of bacillary origin the other malignant infectious diseases are equally so, but we are warned that it will not do to press the analogical argument too far, or to make too large inductions from the facts already in our possession. We have a right to conclude from the data already acquired respecting anthrax, that all cases of this disease wherever occurring are bacillary, but we are not justified from these data in affirming that the bacterial nature of the *materies morbi* of typhoid fever, diphtheria, scarlatina, etc., is proved. We can only say that such conclusion is very probable, in fact more probable in the present state of science than any other. But for each specific disease the problem of causation must be worked out independently, as has been done for anthrax.

It is needless to say that the work is being pushed with vigor, and that what a late writer has been pleased to call the "bacteria mania" has taken possession of pathologists everywhere. It is scarcely three years since we were first startled by the announcement that the microbe of tuberculosis, whose infectiousness had been established since Villemin's conclusive experiments—that the microbe on whose existence this disease depends and without which no such morbid formation as tubercle is possible, had been found, cultivated, and its causal relation to tuberculosis proved according to the most rigorous scientific methods by Dr. Robert Koch, a German physician. So convincing was the demonstration (confirmed as it has been since by numerous observers) that the majority of scientific physicians the world over have given their adhesion to the new etiological doctrine, and those who still remain sceptical as to the causal connection between

the microbe of Koch and tubercle, admit the general concomitance of the two, and the diagnostic value of the presence of the bacillus. Though almost the last discovered, the tubercle bacillus is perhaps the best known of all the bacteria; it readily stains with aniline and is not decolorized by nitric acid; it thrives in the bodies of warm-blooded animals when inoculated, producing irritation, inflammation and the formation of tubercle granula; it does not live in the air at the usual temperature but requires a temperature approaching to that of the human body; its growth entirely ceases below 82° F. and above 107° F., and it does not form spores in the air; it grows slowly, requiring many days for its development; the healthy human body seems to offer an invincible resistance to its invasion, but in certain diseased conditions of the respiratory passages of a catarrhal or inflammatory nature, with stagnation of the air and imperfect aëration of the alveoli, and there is still, perhaps, an unknown factor which we must designate as a want of inherent vitality in the pulmonary cells—it finds a suitable habitat, grows and luxuriates, constituting that destructive disease of the lungs with which we are all so familiar, and from which recoveries are so exceptional. The bacillus forms spores while in the lungs; these, like spores in general, possess an extraordinary vitality; they impregnate the sputa of the phthisical patient, and becoming dried, and forming dust with the muco-purulent products of expectoration, pervade the apartments and dwellings of tuberculous subjects. No one has done more to elucidate this subject than Professor Germain Sée, whose work on Bacillary Phthisis of the Lungs, published last year, is a profound study of this disease from the standpoint of the new etiology.[1] In this treatise the history of tubercle is preceded by a history of microphytes in general, then of the specific microbe known now as Koch's bacillus; the relationship of the bacillus to the "predisposing causes" is pointed out, as well as its diagnostic and prognostic value in all stages of the disease; in fine, the prophylaxis and treatment of phthisis are shown to derive a special illumination from the new doctrine.

The present volume continues the subject of the infectious diseases of the lungs, being occupied with such as are not tuberculous. The bronchi being appurtenances of the lungs, and the graver bronchial inflammations

[1] It was intended to have made this book a part of the present series, but the labor of translation, while in progress, was interrupted by the intelligence that the work had already been translated in England and was on the point of being published.

always involving the alveoli, it was both convenient and proper to consider the bronchites under the head of pulmonary maladies. It is true that simple acute bronchitis is not a microbiotic disease, but the severer forms of bronchitis are—whether occurring primarily, as influenza, which is due to an atmospheric ferment, or secondarily as a sequel of such acute infectious diseases as measles, small-pox, or pertussis. It cannot but be a surprise to the medical Rip Van Winkle to be informed that acute pneumonia is dependent on an atmospheric infection, and that under certain circumstances (unlike influenza) it has been propagated by contagion; the recent researches of Friedländer and Talamon go far to show that this common malady is a specific affection due to the localization in one or both lungs of a microbe whose presence and depredations are the cause of the subsequent constitutional symptoms; in fact these experimenters claim to have discovered the microbe of lung fever, to have cultivated it and to have reproduced in animals the original disease with its characteristic symptoms and lesions.

Some may be disposed to doubt whether *cancer* be an infectious disease, though it certainly is hereditarily transmissible; analogy, however, would class it by the side of tuberculosis and insist on a similarity of causation. The section devoted to pulmonary cancer seems to me especially valuable.

Syphilis is the type of infectious diseases which are almost exclusively human; zealous pathogenists will always continue searching for the microbe of this disease till they find it. The chapter on pulmonary syphilis will be read with interest and profit.

Gangrene of the lungs is infectious in so far as it is caused by the microbes of putrefaction; the active agency of septic bacteria in this disease seems proved beyond any reasonable doubt. The chapter on pulmonary gangrene is quite complete, as is the final chapter on Hydatids of the Lung, an affection with which fortunately American physicians will very seldom have occasion to deal.

In the portion of the work devoted to Therapeutics, Prof. Sée labors to make the new views of causation do practical service, and if the old antiphlogistic and contra-stimulant methods of treatment were not already dead past resuscitation, they could no longer be defensible.

Whether the therapeutic application to the human infectious diseases of a wider and more accurate acquaintance with the special morbific agents,—supposing it to be finally proved that the *materies morbi* of all these diseases be a *contagium vivum*—shall verify the old adage "knowledge is power,"

it remains for the future to decide. It is not too much to expect that such will be the case; that as we come to understand the conditions under which certain maladies arise, we shall be able to master those conditions. "Nous vivons dans un temps où il est bon de vivre quand on s'intéresse aux choses de la médecine" (we live in a time when it is good to live for one who is interested in the things pertaining to medicine), says Prof. Bouchard.

Professor Germain Sée is one of the live men of the profession who has done much good work in clinical and experimental therapeutics. He is a veteran in practice; having been over forty years doing hard professional labor; besides the care of a large private *clientèle* in Paris, he is physician to the Hôtel Dieu and a popular clinical teacher. He is author of a number of excellent medical works, among which I may specify his "Pathologie Experimentale," now long out of print, his "Maladies du Cœur," which has passed through two editions, and his "Dyspepsies," a masterly work on the indigestions, which appeared in 1883. The present work is shortly to be followed by another on the "Acute Infectious Diseases, and Fevers."

NEWBURYPORT, MASS., *June 25th*, 1885.

By kindness of his Paris correspondent, Dr. Dujardin Beaumetz, the translator is permitted to present a valuable article on Bacteria as an appendix. Dr. George M. Sternberg, of world-wide reputation as a mycologist, has also contributed, as Appendix B, a reprint of his recent important review article (Am. Jour. Med. Sciences, July, 1885) on the "Pneumococcus of Friedländer." Dr. Sternberg's studies may or may not be regarded as having confirmed the main thesis of the author of the chapter on Pneumonia; they certainly have opened up a most interesting line of inquiry as to the possible morbiginous influence, under certain conditions of depressed vitality, of bacteria which are occasional components of healthy animal fluids.

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DISEASES OF THE LUNGS

OF A

SPECIFIC BUT NOT TUBERCULOUS NATURE.

INTRODUCTION.

This title clearly indicates the nature of the broncho-pulmonary maladies which are the subject of our present clinical studies. The specificity is the result of but one cause, a living agent, parasitic or virulent, and is the characteristic not only of tuberculosis (which is bacillary in its origin) but also of the greater part of the acute broncho-pulmonary diseases, particularly certain bronchites, the pneumonias and gangrenous affections of the lungs, which form a first series, easy to define and justify. Certain chronic diseases have the same distinctive quality; these are syphilis, the verminous affections and cancer, which without being virulent is at least auto-infectious.

The specificity of this second series of diseases has long been recognized.

FIRST SERIES. ACUTE SPECIFIC DISEASES.

Bronchites, Pneumonias, Gangrenes.

We find in this morbid group the acute pulmonary diseases which have always passed for types of the most clearly pronounced phlegmasias. But clinical experience based on the recent discoveries of microbiology, does not allow the least doubt as to the parasitic origin of most of the bronchites, of all the pneumonias, of all the gangrenes.

The Bronchites.—The specific bronchites are some of them contagious, like the rubeolic catarrh, others epidemic, like influenza. Re-

sembling by their anatomical characteristics simple bronchitis from cold (ordinary bronchitis), they are distinct in their origin and etiology, and this suffices to class them among the infectious diseases; but this is not all Even the simple forms of bronchitis when they invade the capillary bronchial twigs, when they become transformed into broncho-pneumonia, catarrhal pneumonia, peri-pneumonia notha, take on manifestly the parasitic character, and from this important standpoint cannot be distinguished from the pneumonias so called.

Infectious Pneumonia.—The infectious pneumonias are those which are called acute by reason of their inflammatory nature, fibrinous in consequence of the intra-alveolar exudation characteristic of the inflammation, lobar on account of the extension or intensity of the phlegmasia.

Considerations, to be afterward mentioned, justify us in henceforth classing these common forms of pneumonia (lung fever) in the category of infectious diseases. They ought then to be regarded in their true light by the physician and no longer be supposed tributary to the treatment called anti-phlogistic, or, more properly speaking, spoliative, debilitating, contra-stimulant. In fact the therapeutics of pneumonia will in the future be that of all the infectious diseases, it will comprehend, especially in the absence of effective anti-parasitic medication, the means proper for supporting the strength of the patient and enabling him to struggle against the infectious agent and its deleterious and lethal effects.

Gangrenes.—Gangrene also used to be considered as the result of inflammation *in excess;* it becomes to-day a disease of putrid infection, due to septic microbes that prey on the organized tissues. The above comprehends the morbid processes of the acute stage.

SECOND SERIES. CHRONIC SPECIFIC DISEASES OF THE LUNGS.

The second series comprises three groups of diseases which habitually pursue a chronic course and whose specificity is not disputed. It comprehends syphilis and cancer of the lungs and the verminous (hydatid) affections.

Syphilis.—Although the micro-organism of syphilis has not yet been found, syphilis none the less constitutes the type of virulent, infectious, and inoculable maladies. It spares no part or element of the respiratory organs; it invades under the most diverse forms larynx and

trachea, bronchi and alveoli. It is this aggregate of syphilitic lesions which is included under the name of pulmonary syphilis.

Cancer.—Cancer has not yet been proved to possess the property of virulence, it is neither parasitic nor contagious, but it is auto-infectious, and this is why it is regarded as a malignant production. Its legitimate place seems to be by the side of syphilis, although unfortunately separated from the latter by its widely different and confessedly unsatisfactory therapeutics.

Hydatids.—Hydatids and mycosis of the lungs form the natural completion of the parasitic diseases. The first manifest themselves as tumors, often accessible to surgery; the second is still a subject of investigation.

To sum up, the grouping together of all these diseases need cause no surprise. They are separated only by their anatomical characters, and this point of view concerns us the least; we are chiefly interested in them by reason of the similar etiological conditions under which they all originate, and pathogeny compels the classification which we adopt. Medicine in the future will cease to have an anatomical basis, for it will be founded on etiology.

FIRST SERIES.

ACUTE SPECIFIC DISEASES.

PART I.

CHAPTER I.

ACUTE BRONCHIAL INFLAMMATIONS.

Parallelism between Catarrh and Inflammation.

Formerly the view of Traube was prevalent which admitted as catarrh only the affection of the mucous membrane characterized by a secretion rich in mucine and poor in cellular elements, the latter originating only in the superficial layers of the membrane. If this view were correct, catarrh would be comparatively rare. What clinical experiences teaches us is directly the contrary, namely, that the secretions or exudations are rich in histological elements which are found throughout the entire mucous membrane which is the seat of irritation. The morbid process which may be properly called catarrhal inflammation, especially when considered from an anatomical point of view, is characterized not only by augmentation of mucus on the part of the affected membrane, but also by the fact that the latter presents all the characteristics of true inflammation. The proof that it is so is, as Riegel observes[1] found in the condition of the sputa. which are distinguished by their great richness in mucine, on the one hand, and on the other by the presence of numerous cellular elements, especially lymph corpuscles. These two factors vary in relative proportion according to the different stages and forms of the catarrhal inflammation; sometimes the anatomical lesions predominate, to wit the hyperæmia and swelling of the mucous membrane which constitute the phlegmasia; sometimes the most prominent symptom consists in the production of an excessive secretion of mucus, of serum or of pus, and you have the mucous, pituitous, bronchorrœic catarrh of Laennec. These distinctions appertain only to chronic catarrh, or at the most to the terminal periods of acute catarrh; but in reality the question of chief importance is not concerning the nature or the quantity of liquid secreted, but rather concerning the nature of the anatomical lesions which are the origin of the pathological secretion.

What are then the inflammatory lesions? Is it a matter of simple hyperæmia? We know that in the case of glands the secretion is always accompanied with an increased afflux of blood at the moment of functionating; when the labor of the gland is over the blood flow is reduced to a minimum; the work of an organ then necessitates more blood, but the congestion thus resulting is temporary, and never leaves any changes discoverable after death. Have we, on the other hand, to do with a real inflammation of the mucosa? If this be the case there are always three phenomena indissolubly associated, viz., dilatation of the blood-vessels, retardation of the blood current, diapedesis of the leucocytes, and the latter can take place only when you have the two states of the circulation just

[1] Ziemssen's Cyclopædia (Wood's Ed.), vol. iv., p. 298.

given. The diapedesis is the essential character of the inflammation of which the ectasy of the blood-vessels is the condition, and the hyperæmia the prelude.[1]

Now hyperæmia plays the same part in the evolution of the morbid process, whether it be active or passive. In the venous stases resulting from troubles of the cardiac circulation, the mucous secretion is quite as abundant as when it results from arterial congestion. If the hyperæmia passes on a stage farther, the swollen mucosa and parenchyma become the seat of more profound modifications; there is now inflammation manifesting itself by its tokens—stagnation of red globules, migration of leucocytes, transudation of serum, fibrine and albumen. The circulatory system subserves these morbid changes by visible alterations, along with dilatation of the vessels, and slowing of the blood current.

If this be so, if the extravasation of histological elements forms the basis of the phlogosis, the swelling of the morbid tissue is easily explained. The inflamed mucosa also undergoes tumefaction, and if the bronchus be of small calibre it may become so much constricted as to give rise to the most grave consequences, such as atelectasis or collapse of the corresponding lobules; the larger bronchi, on the contrary, may undergo considerable intumescence without any impediment to the ingress of air being thereby occasioned.

Hyperæmia in the bronchi is opposed by another cause which resists dilatation of the vessels and swelling; I refer to the abundance of elastic fibres in the mucous membrane which antagonize vascular distention, often effectually overcoming blood pressure, and preventing the disastrous results which would otherwise follow; we see in this moreover an explanation of the fact that where swelling has existed during life there is sometimes no manifestation of it after death. The case is different with catarrhal affections which have lasted a long time. The epithelial cells undergo a marked increase, and there are degenerative alterations which may even gain the sub-mucous connective tissue as well as the glandular apparatus, so as to cause hypertrophy of the entire mucous membrane. When this process takes place we have no longer merely hypersecretion: the secreted mucus is profoundly altered, being now an epithelial leucocyte and purulent production.

The Catarrh is not a Simple Hypersecretion.—We have not then simply increased secretion, without the participation of the vascular and cellulo-glandular elements; there is always hyperæmia, diapedesis of leucocytes, and degeneration of the histological substrata. This then is the congeries of lesions comprising the complex condition called inflammatory, of which no one lesion has any value or significance apart from the others. In fact the hyperæmia may be merely functional, non-inflammatory and of transient duration. The degeneration of the fixed cells belongs to certain regressive states which pertain to the terminal periods of inflammation. The hyperæmia distinguishes the acute catarrhs, structural degenerations the chronic. The latter form a distinct class, to be hereafter considered.[2]

[1] The discovery of the diapedesis, or passage of the white corpuscles through the dilated capillaries, denied recently by Wharton-Jones (Lancet, October 11th, 1884), wrongly attributed to Cohnheim and Waller, really belongs to Dutrochet, who very clearly and fully described the emigration of the leucocytes in a work published in 1824 entitled "*Recherches anatomiques et physiologiques sur la structure intime des animaux et des vegetaux.*"—Trans.

[2] i.e., in a volume which Prof. Sée is now preparing.

CHAPTER II.

ETIOLOGY OF THE ACUTE BRONCHO-CATARRHS.

Etiological Divisions.

The acute broncho-catarrhs may be grouped into four classes.

Accidental Broncho-catarrh, or Bronchitis a Frigore.—There exist certain acute catarrhs which are due to the topical effect of cold air on the mucous membrane of the air passages or on the skin.

Primitive Infectious Broncho-catarrhs.—Another division, interesting from a different point of view, though less frequently met in practice, comprehends the catarrhs of microbic diseases which invariably localize themselves in the bronchi and lungs; these are: (*a*) Influenza, which is a disease essentially epidemic. (*b*) Whooping-cough, which is manifestly contagious and is not recurrent; (*c*) Measles, which is both contagious and epidemic. The bronchial catarrh is an essential phenomenon of all three diseases.

Broncho-catarrhs that are Secondarily Infectious.—Certain infectious diseases without being essentially broncho-pulmonary, may likewise invade the respiratory organs and determine there a true catarrhal inflammation. Such are *small-pox*, which often occasions pustulation and consequent inflammation of the larynx, trachea, and bronchi; *typhoid-fever*, which is never attended with catarrhal irritation at its onset, as is measles, and never produces broncho-pulmonary lesions except as the result of general or cardiac adynamia, that is to say by the intermediation of a passive congestion of the lungs, the antecedent of the bronchial hypersecretion.

This is the place to mention the cachetic and septic forms of bronchitis, which, supervening in the extreme periods of cachexia depend on the general enfeeblement, on the difficulties in the way of expectoration which exist in these cases, and the consequent accumulation and disintegration of sputa which have become putrid and are susceptible of absorption.

Bronchitis of Physico-chemical Origin.—A fourth and last class of bronchites has its origin in local irritants which, after the manner of deleterious gases or particles of dust in the atmosphere, penetrate the air passages. Various chemical irritants, which after having been absorbed are eliminated by the lungs, may, like iodine and bromine, act indirectly on the mucosa and provoke the development of a catarrh; either, it may be, sanitary, like the iodine catarrh, or grave like the bromide bronchitis, which when it occurs constitutes a real impediment to the bromide medication.

Etiology of Common or Meteorologic Bronchitis.

If there still remains a disease which may be rightly attributable to thermal influences, it is assuredly common bronchitis. Till a very recent

period, cold was regarded as causative in all inflammations; the microbiological researches of the last few years have assigned to the general dogma of diseases *à frigore* its proper place. The more important of them, the pneumonias, pleurisies, the bronchites, as a whole, even rheumatism, have passed to the rank of infectious diseases; there remains but one acknowledged to be amenable to cold alone, namely common bronchitis, or rheum of the air passages. The various factors which enter into the constitution of a climate, to wit, cold, heat, humidity, winds, may by their unpropitious combination, occasion the development of bronchial catarrh. The atmosphere, with its frequent oscillations of cold and moist temperatures and traversed by raw wind currents, presents conditions which are potent for mischief to the respiratory mucous membranes.

Climates and Regions that favor Bronchial Catarrhs.—According to the researches of Hirsch, the farther one is removed from the equatorial zone towards the north, the more the conditions abound which generate respiratory catarrhs. It is in the cold or temperate, and at the same time damp regions, which present the most frequent, sudden and intense oscillations of temperature, that catarrhal affections predominate. This is the general law. There is besides a special law which regulates the relations of respiratory diseases with the variations of the seasons; the catarrhs have their maxium of prevalence in countries within the same parallels of latitude during seasons, months, and years, marked by great hygrometric fluctuations; this is especially true for periods of the year when temperature and dew point fall closely together; in which the atmosphere, saturated with water, is continually moist, periods which with us occur especially in the spring and fall.

Action of Cold on the Individual.—Sudden changes of temperature from hot to cold and *vice versa;* cold winds abruptly succeeding a season of warmth, and laden with moisture, furnish occasions the most productive of chilling to the human organism, and the consequent development of broncho-catarrhs. But how is the irritation of the mucous membranes brought about? Here we must appeal to physiological experimentation.

When, after the example of Riegel, Ackerman and of Rosenthal, you remove an animal from a heated box to an ordinary atmosphere, its temperature falls not only to the normal, but below it. This is the explanation: By reason of the elevated temperature the blood-vessels dilate, whether by paralysis as Rosenthal thinks, or actively by excitation of the vaso dilators: there is an afflux of blood to the skin, and the animal loses so much heat by radiation that its internal temperature can for some time rise but very slightly. When he is brought into a cooler room the blood-vessels still remain dilated a certain time, proportionate to the period in which he remained in the over-heated atmosphere. Now that there exists a marked difference between the heat of the animal and that of the surrounding medium, the animal must lose much more heat than an animal in the normal state when placed in the same apartment, and this is in fact what takes place.

Clinical Remarks.

Sudden Transit from Warm to Cooler Air.—Acute catarrh does not ensue as a consequence of a gradual transition from a higher to a lower temperature, say from 86° or 90° F. to the ordinary temperature of the air; it is necessary that this transition be sudden. When the body is overheated the superficial vessels are enormously dilated; if it be then sud-

denly exposed to the cold, the multiplied contact of the blood with the surrounding cooler air determines an immediate loss of caloric, and the cooled blood courses through the internal organs, reducing the heat of these parts more completely and more suddenly than would have been the case if the body had been simply exposed to the cold without the previous heating. This instantaneous cooling of the internal viscera may be a real injury to them, and disorder their functions; we do not know in advance what shall be the organ affected—we are told that it is the *locus minoris resistentiæ* (place of least resistance), but this is simply the expression of a fact. We know, too, that the same organ in the same patient after having once undergone the catarrhal perturbation is more ready to suffer repetitions of the attacks from thermal disturbances.

Action of Cold Winds.—The same effect is experienced when a cold draught or chilly wind comes in contact with the surface of the body at a time when it is bathed in sweat; there is rapid and excessive loss of caloric from the periphery, and the temperature of the deeper parts is lowered beyond the normal mean.

Action of Cold Water on the Body when Perspiring.—It is the same when profuse perspiration is checked by the application of cold water to the tegumentary parts, whether this be by means of a shower of rain, or an atmosphere saturated with moisture; you have here a similar abrupt transition from an overheated to a cold medium.

Therapeutical Consequences.—As a prophylaxis against this exaggerated impressionability of the peripheral vessels we have a wonderfully invigorating mode of treatment in hydrotherapy, particularly in cold douches. By the sudden and violent effect of these cold applications to the cutaneous surface, the peripheral vessels undergo a marked constriction, and the integument is strengthened and made more resistant against refrigerant influences, a state of permanent tonicity of the vessels of the skin being determined; by no way is the individual more effectually toughened against colds. It is known, moreover, that douches of from 60° to 68° F. administered from three to seven minutes to a healthy man, not only do not lower the axillary temperature, but they slightly elevate it;[1] hence without any interruption to normal thermogenesis they fortify the peripheral vascular system. This may be regarded as proved, as well as the good effects of hydrotherapeutics in diminishing or destroying the susceptibility of the bronchi. I insist on this fact, which is not applicable to the bronchites called pretuberculous,—if such exist—in which case hydrotherapy would be attended with bad results. I do not, however, believe in this pretuberculous bronchitis; Laennec says, as I think truly, "There is only simple bronchitis, and tuberculosis with bronchitis."

Predisposing Causes.

Numerous have been the predisposing causes of bronchial catarrh imagined by authorities; causes pertaining to the constitution, temperament, sex, age, even corpulence of the individual; for many of these there seems to us to be little foundation.

These catarrhal affections have been said to be the peculiar prerogative of all those who possess a minimum power of resistance, innate or acquired, to meteorological influences. As it is impossible to compute the sum of

[1] Experiments of Labade-Lagrave, Liebermeister, etc.

the vital energies, the proposition has been stated somewhat differently, and certain lymphatic conditions, scrofula, anæmia, and even a disposition to obesity, have been accused as factors in the prevalence of catarrhs. It is, however, a matter of common observation that not all debilitated or scrofulous persons are subjects of catarrh, and as for those who are such, we have always to invoke a *locus minoris resistentiæ, i.e.*, an unknown constitutional defect—the individual resists, but the bronchi yield, we know not why. The further consideration of this subject properly belongs to the head of chronic catarrhs and relapsing bronchitis and will be fully discussed in another volume.[1] The rheums (rightly so called), preferably attack those who are the most exposed by their occupation to "catching cold;" this is why men are more subject to acute catarrh than women—sex alone has little influence as a predisposing cause.

Age, on the other hand, and especially infancy, has considerable influence, but here, too, much depends on whether the infant be well cared for or exposed to the cold. The newly born, well protected by their wrappings seldom take cold; from six months and onward catarrhs are frequent. According to Giegil comparatively more legitimately born children die of respiratory diseases in the first year of life than illegitimate children; the parents of the former are too scrupulously careful to preserve their offspring from every breath of cold air, and use excessive precautions; the result is that these infants readily become debilitated and enervated, deriving detriment from the least change in the weather. On the other hand, little illegitimates that do not receive such tender care and attention become toughened against the inclemencies of their surroundings; they are like individuals whose superficial vessels have acquired by hydropathy the tonicity requisite to resist the vaso-dilator or paralyzing action of warm air on the tegumentary circulatory system.

As for the influence of dentition, it has been remarked, and with truth, that infants are more subject to catarrhal affections of the air passages during teething than before or after, getting rid of their rheums when the teeth are cut. But the relationship of bronchial catarrh to the physiological functions of dentition is not so sharply defined that one can tell these teething catarrhs from other catarrhs, the effect of cold, by which the infant may be attacked at the time of dentition. The bronchitis which is neglected because supposed to be physiological, may readily, owing to closeness of the tubes, take on the capillary or pneumonic form,—to which infants are especially liable as a sequel of ordinary bronchitis. In older children or adults, on the contrary, the pneumonia is more likely to be primitive than secondary.

On the supposition that teething has an influence in the genesis of bronchitis, what is the *modus operandi?* It has been alleged that the pain attending the development and eruption of teeth enfeebles the infant, who is thereby more susceptible to the action of cold and vicissitudes of the weather. This is the popular view. It is not, however, true that children as a rule suffer pain during the period of dental evolution. Nor does there seem to be much reason in the theory that the physiological labor of dental ossification is attended with constitutional debilitation and that the greater impressionability of the child may be thus explained. It is, to say the least, questionable whether such debilitation as an integral part of the development of each of the teeth, really exists. Then again

[1] Vol. III. of the French series.

the view has been put forth that coincidently with the eruption of each tooth there is a general determination of blood to the mucous membranes, gingival, nasal, laryngeal, bronchial, hence the rheum. This hypothesis may well find a place in the category of congestive diatheses, but it finds no support in clinical medicine. Again, reflex irritation has been invoked, the point of origin being the terminal dental nerve, and the cough of teething has been compared to the famous gastric cough, and the verminous cough, concerning which there is still dispute. This physiological theory, according to which the inflamed gums start an impression which goes to the bulb and is reflected back on the motor expiratory nerves, giving rise to cough,—would be the most reasonable if it could not only explain the motor mechanism of the cough but also the secretory mechanism of the bronchial catarrh. These problems, instead of being solved, have hardly even been discussed, and yet the therapeutics of these catarrhal teething coughs cannot be established till their pathogeny is known; under the head of treatment we shall allude to this subject again.

After the Age of Five Years the tendency to catarrhs goes on lessening; it falls to the minimum in adolescence and the adult age, to rise to the maximum in old age. At this closing period of life, it is correct to say that the bronchial catarrh is only a secondary result, *i.e.*, the effect of venous stases in the lungs, and it will not do to confound these passive symptomatic, consecutive hyperæmias, with the active inflammatory fluxions which prepare the way for bronchitis.

CHAPTER III.

ETIOLOGY OF THE SPECIFIC CATARRHS. INFLUENZA.

Among the primarily infectious catarrhs we shall study only influenza; the catarrhs attending measles and whooping-cough will find their respective place in the treatise on acute infectious diseases.

Etiological Definition.—Influenza ("La Grippe" of French pathologists, from the word *agripper*, to seize) is a disease which is epidemic, non-contagious, independent of thermal conditions, and apparenly due to an atmospheric agent. It is characterized essentially by catarrhal irritation or inflammation of the respiratory organs.

Influenza is Epidemic and Non-Contagious.

Apart from epidemics influenza does not occur. Ordinary catarrhs may be prevalent in certain seasons in certain countries, but they never have the universal diffusion which distinguishes the disease under consideration; while presenting certain semeiological resemblances to influenza they can always be recognized, and when the epidemic commences by isolated cases, it very soon unmasks itself and gives the accompanying catarrh its true character. Since the year 1510 commenced a series of epidemics whose extension has never been equalled by any known infectious disease; more than ninety epidemics have been reckoned, which have invaded entire countries, often wide latitudes representing a considerable portion of the habitable globe. In each locality invaded, in each populous city it generally breaks out at once in such a manner as to smite simultaneously thousands of individuals; rarely, as in the case of the famous epidemic of 1836–1837 which prevailed in St. Petersburg and Paris, the disease confines itself to certain quarters of the town. This diffusion is not only proof of its epidemic character, but its rapidity of propagation seems irreconcilable with transmission by way of contagion. In vain during these epidemics one might sequester one's self or remain within doors, it was impossible to escape the disease, whose infection would penetrate apartments the most close, and attack persons the best protected against atmospheric influences. By whom, or by what is the contagion imparted? It is not by the patients nor by their clothing or expectoration. You will see crowded tenements, barracks, schools, workshops, factories which, while freely communicating with the city where the influenza is raging, may present only sporadic cases or escape altogether. On the other hand, ship crews have been invaded when far out at sea and deprived of all communication with the land. In the one case contagion was impotent, in the other impossible.

These facts prove that the transmission is not from individual to in-

dividual, and that it can only take place by the medium of the atmosphere; the diffusion and explosion of the disease are explicable in no other way.

Influenza Apparently of Parasitic Origin.

It is easy to show that this disease is independent of climatic conditions as well as the regular variations of the atmosphere; it is not in fact amenable to any of the common causes which produce bronchial catarrh. It prevails in equatorial countries as well as in the cold northern regions; for instance, the epidemic of 1580 spread from the south-east to the north-west, from Constantinople to Hungary, and from Venice to Germany, where it attacked half the population, then it reached Norway and Russia to return in the summer to Spain and Italy, where it caused, especially in Rome and Madrid, a considerable mortality, which Wier attributes to the employment of blood-letting. Hence latitudes and climates go for nothing as factors of causation. Nor do seasons have any more influence; you would naturally expect increase of the epidemic during cold seasons, but this is what the statistics of 1837 give:—In January, coming from the north it made its appearance in London, where it attacked in a severe form almost the entire population; then it passed over to Germany and France; at the beginning of February all Switzerland and France were invaded, in March it had reached Spain. This same epidemic spread from the meridional continents of the globe, raged at Sidney (Australia) and the Cape, and in the same season and about the same time that it visited the north of Europe, it prevailed at the antipodes during climatic conditions the opposite of those existing in Europe during its prevalence there. Seasonal and diurnal variations of temperature, barometric or hygrometric oscillations, the influences of great atmospheric currents, have no effect on the production, the march, or the evolution of these epidemics, which in general last several weeks and sometimes appear in a very benign form, sometimes in types of the gravest character. Although as a general rule the direction of epidemics is from the east to the west, accompanying east or northwest winds, it is no less true that there are numerous exceptions to this rule, and that in general the march of these epidemics is wholly indifferent to ordinary causes as well as to the various conditions of age or of constitution.

Unable to find the causes of influenza in thermal or hygrometric variations of the air, authorities have assumed a causal relation between a highly ozonized condition of the atmosphere and this disease. The ozonometric state of the atmosphere noted by Granaro during the epidemic of Genoa in 1858 seemed to become aggravated with the rise and progress of the epidemic, but no one has been able to prove the least correlation between this disease and the degree of ozonization of the air, although an atmosphere highly charged with ozone is doubtless an important factor in the causation and frequency of bronchial catarrhs; as far as influenza is concerned it would seem that ozone must constitute an excellent means of destroying the parasite, if the latter affection be of microphytic nature.

After having demonstrated the independence of all atmospheric changes, it remains to prove that the disease in question is due to a specific agent diffused in the atmosphere, miasm or microphyte. If the causal agent has not yet been found, if it has hardly been sought for, it is

because the disease is generally benign, and without serious consequences, and because epidemics have not now prevailed for many years, certainly not since the study of microbiology has engaged the atention of enlightened physicians the world over. But enough is known of the *materies morbi* to warrant the conclusion that it is not of telluric origin, and that it has no affinity with the miasm which constitutes the essence of marsh malaria.

Graves, Jaccoud, and especially Biermer, without specifying the nature of the morbid agent, attribute to it an indigenous origin. Biermer bases his opinion especially on the fact that certain localities, certain territories, certain streets and houses seem always and in each epidemic to be more invaded than neighboring localities or habitations; the former become little epidemic centres which by their multiplication and union constitute a focus of irradiations for a whole country or continent. But it must not be forgotten that this centralization is uncommon; the epidemic generally smites the greater part of the population in divers points and places wide apart, selecting preferably cities and thickly crowded localities; it bursts upon a region, an entire country, like a tornado; and the theory of little centres of origin, telluric foci, widely separated, gradually meeting to accomplish their spreading devastation, finds little support in fact.

Comparison with the Marsh Microphyte.

Among the diseases which have a telluric origin the most clearly defined, the best proved is malaria. The specific microphytes of this malady, long suspected, and of late recognized and clearly pointed out by Tomasi-Crudeli, Klebs, Marchiafava, and Perroncito, constitute what are called miasms. They originate in the soil, that is to say in paludal lands where there is much decaying vegetable matter and where multitudes of morbiginous microphytes undergo development; but with these peculiar characters, that their germs become diffused through the air only to a limited extent, do not rise above a certain height and are confined principally to the lower strata of the atmosphere. Malarious diseases are, then, endemic, and very exceptionally epidemic. We cannot, therefore, compare influenza to paludal fevers nor the hypothetical parasite of the former to the clearly proved micro-organisms of malaria.

There are only these two diseases, influenza and malarious fever which propagate themselves exclusively by the atmosphere. But while the one has a certain telluric source the other cannot possibly be said to derive its sole origin in this way, for it is not autochthonous, it has no native country, it arises indifferently in any one of the five quarters of the globe and there propagates itself across the sea to all the countries of the old or new world, being essentially pandemic in its character.

Therefore there is nothing telluric about it; everything leads logically to the conclusion that this virus must be referred to an atmosphere vitiated by a living organism which multiplies and propagates itself ad infinitum, which affects all individuals without regard to age or constitution, smites them suddenly, without any period of incubation, then disappears after a period of from four to eight weeks of prevalence, and does not generally come again till after the lapse of several years. For more than ten years influenza had not been heard if in our country when Mathieu, my clinical chief, and myself observed an outbreak of it in our midst; this was about the end of 1883; the epidemic appeared in a grave form and lasted about two months.

CHAPTER IV.

SEMEIOLOGY OF SIMPLE ACUTE BRONCHITIS.

Instead of giving a complete description of catarrhal bronchitis, I shall consider only the leading phenomena, which are: the cough, the expectoration, the râles; the other symptoms, such as dyspnœa and fever, have more importance in prognosis than in diagnosis and will come up later for study.

Signs and Symptoms of Acute Bronchitis.

Bronchitis, whatever may be its origin, passes through a regular evolution, designated anciently under the name of periods of crudity and of coction; to each of these states corresponds a profound modification in the manifestations.

Cough.—The cough is the first and most important of functional troubles.

Period of Crudity.—After repeated chills, after a moderate elevation of the temperature, some degree of prostration with loss of appetite, a cough makes its appearance, which is at first dry like the bronchial mucous membrane itself, sometimes moderate, sometimes painful and accompanied by a sensation of tickling or of smarting in the windpipe; this is called irritative cough. There are normally in the respiratory passages certain tussigenous zones of varying potentiality; these are more especially the interarytenoidal fissure, which is innervated by the superior laryngeal nerve, the posterior aspect of the trachea, the mucous membrane at the bifurcation of the trachea, and the bronchial tubes themselves, which are supplied with nerves from the terminal filaments of the pneumogastric; the recurrents here take no part.[1] But in

[1] *Tussigenous zones or regions.*—The cough, taking its point of departure in a sensory impression, has for its field of origin the entire territory of the vagus. The series of tussigenous zones which provoke this kind of forced, resistless, painful and noisy expiration are as follows:

1. *Laryngeal Mucosa.* The laryngeal cough has its nervous source in the superior laryngeal, which provides the laryngeal mucosa with sensory nerves. But, as Rosenthal has shown, the facility of provoking this cough is not equally possessed by all portions of this mucous membrane. Nothnagel, by experiments on dogs, has shown that mechanical irritation only provokes cough when it acts upon the inferior vocal cords, and from thence on the mucosa as far as the cricoid ring of cartilage; the interarytenoidal region should be added. These facts have been verified in man by R. Meyer, who remarks that when certain spasmogenous zones have produced cough, the irritation may gain the neighboring indifferent zones.

2. *Tracheal Mucosa.* The tracheal mucosa is excitable throughout its whole extent. Nothnagel believes, nevertheless, that a stronger irritation is needed than that which provokes the laryngeal cough.

3. *Bronchial Mucosa.* All parts of the bronchial mucosa have not, according

the pathological state the inflammation may give rise to a hyper-excitability of the nerves of the entire bronchial mucosa; from the seat of the catarrhal congestion, especially if it affects the origin of the bronchi, goes a centripetal impression which influences the respiratory centre in the bulb and is reflected on the expiratory nerves in such a manner as to call forth the vehement and noisy expiratory effort called cough, the maximum of which corresponds with the maximum of catarrhal irritation of the most sensitive regions. But there are still other reasons for this neuro-motor hyper-excitability; the more thick and viscous and adherent the exudation the more the efforts of expulsion are difficult, painful and repeated. The cough increases also by the horizontal decubitus, which causes the mucus to accumulate in the excito-motor parts of the air tubes, where the secretory hyperæmia tends also to become exaggerated by this prone position. Moreover the excito-motricity is more pronounced in children, in females and in impressionable individuals, in whom the cough is most obstinate and most persistent.

During the first few days the bronchitis is accompanied by retrosternal pains, sensations of smarting or of tickling along the respiratory passages, twinges shooting along the sides of the chest and toward the epigastrium; all these phenomena cease as soon as the cough becomes looser, which takes place when the stage of coction appears.

Stage of Coction.—The maturation of the exudation makes itself manifest by the more liquid condition, the greater abundance, the lessened consistency of this secretion. The cough now, as it were, finds something to remove, it becomes easier and at the same time seems exercised to some good purpose, readily eliminating the exudation; the air expired by coughing in penetrating and removing the sputa, communicates to the cough, which before was harsh, a fuller, graver tone.

Expectoration, Initial Phase.—At the onset of the disease the expectoration is very trifling in quantity and consists of a frothy transparent mucus, colorless, or grayish white, poor in cells, of almost gelatinous consistency, so viscous as to remain adherent to the bottom of the vessel, even when the latter is inverted. From a histo-chemical point of view this mucus is composed of mucine mixed with a large quantity of water, divers salts and especially chloride of sodium. The microscope reveals only traces of pus and mucous cells with some desquamated epithelium of cylindrical, vibratile and pavement varieties, these latter coming from the upper air passages, trachea, vocal chords, and mucous glands.

Phase of Coction.—Little by little this mucus becomes more rich

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to Nothnagel, the same impressionability. The most irritable region is the bifurcation of the bronchi. At the same time experience proves that in man, the inflamed mucosa is irritable at all points, and that the slightest irritants suffice to provoke cough, no matter from what part they start, and that the reflexly operating hyperæsthesia has its special seat in the inflamed area.

4. *Pulmonary Parenchyma.* The pulmonary parenchyma is of itself incapable of producing this result; the exudations which the cough removes do not provoke expulsive efforts till they reach the afferent bronchi.

5. *Pleura.* According to experiments of Nothnagel on animals, irritation of the pleura is incapable of producing cough, nevertheless a cough is often observed in dry pleurisy and can be provoked by compressing the corresponding parts of the thorax.

6. *Ear Passages, Pharynx.* The irritation may take its starting point from the ear passages and pharynx.

7. *Stomach. Gastric Cough.* The existence of a gastric cough is generally admitted; oftener admitted than proved.—See, Phtisie Bacillaire, p. 169.

in cells, opaque, less viscous, and is covered with striæ and yellow points which consist of leucocytes; thereafter the exudation is less adherent to the mucosa, and we have the muco-purulent sputa (sputum of coction). Finally it becomes quite opaque, abundant and yellow; the cough is now more infrequent but more efficacious to eliminate the exudation products and free the respiration, which was impeded. In children, whatever may be the composition of the exudation, there is no expectoration, the sputa are swallowed.

Auscultation.—The third sign is derived from auscultation; percussion in this disease is negative.

First Period—Dry Rales.—In the first period you note the existence of dry, sonorous, snoring or whistling râles; the first have their seat more especially in the trachea and large bronchi. It has been supposed that the sibilant râles come from the medium sized or smaller bronchi; both kinds of râles ordinarily occupy a large extent and are heard over both lungs. It is important to know the mechanism of the production of sibilant râles, as the treatment depends on this knowledge. The mucous membrane, by reason of the catarrhal inflammation, becomes swollen and thus reduces to some slight extent the lumen of the bronchi, which may then give rise it is said to the sibilance. But as the râles generally occupy the entire respiratory surface, one would have to conclude that the inflammation and the tumefaction involve the whole area of hæmatosis: if this were so, there would be danger of death by asphyxia in the most ordinary cold. We know, moreover, that the swelling of the mucous membrane of the large bronchi is never sufficiently marked to determine any considerable diminution in the calibre of the air tube.

The cause of the dry râles is attributable solely to the passage of the air through the layers of an exudation which is viscous, scanty and adherent to the membrane, and this is why they are heard at the entry as well as at the exit of the tidal wave; and the reason is also plain why the respiratory murmur, especially in inspiration, may be momentarily abolished in the pulmonary lobules, whose afferent bronchus is temporarily obstructed by the exudation. As soon as the mucous plug is displaced by the cough, or by a forced respiration, the silence ceases and gives place anew to sibilance.

Second Stage: Mucous Rales.—As soon as the exudation becomes less glutinous, more loose, more fluid, more abundant, and consequently less adherent to the walls of the air tubes, when, in a word, it is formed of a mucus thick and rich in leucocytes,—marking the second stage of the catarrh,—the râles change their nature and timbre, they become now liquid. When the tidal air detaches the exudation in the form of vesicles or bubbles the rupture of these latter is audible under auscultation as moist râles. These are more sonorous if produced in the large tubes, and if the fluid secreted has considerable consistence, so that vibrations are slowly produced in it. Their volume may even give suggestions as to their origin, and they have hence been divided into coarse large râles, referable to the larger air pipes, moderate sized, or sub-crepitant râles, pertaining to the medium-sized bronchi, and fine sub-crepitant râles, which are supposed to belong to the bronchioles. All these kinds of râles may be heard in the various forms of bronchitis, alternating or varying according to the seat of the catarrh, the requisite condition for their production being that there shall be a thick, mobile liquid exudation in the air tubes, such as the muco-pus of the second stage of catarrh. Their mode of pro-

duction is settled beyond question; it is useless to invoke in explanation the inflammatory swelling of the mucosa. All these râles are due to the passage of the air during inspiration and expiration, through the viscid secretions, the muco-pus of the bronchi. Their mobility and shifting nature prove this; a strong respiration modifies their character; a few efforts of coughing displace them, or cause them momentarily to cease; an emetic, dislodging and removing the bronchial contents, causes them to stop for a longer time.[1]

Secondary Modifications of the Respiratory Murmur.—The râles may even disappear by a quite different mechanism. The accumulation of muco-pus in the bronchi may be so great as to plug up one or more air pipes so as to prevent the penetration of air and formation of bubbles. The bronchial tubes which remain permeable exercise a collateral supplementary function and give occasion to a rude or puerile respiratory murmur, as well as a prolonged expiration.

Dyspnœa and Fever.—To the series of troubles pertaining to the bronchial secretion which manifest themselves by the cough and the râles, we must add dyspnœa (a symptom which, however, is not always or generally present). Two distinct causes determine its presence: the intensity of the catarrh and of the fever. The first cause is of mechanical order: the second, exclusively dynamic, dominates the respiratory troubles to such an extent that in its absence the respiration remains tranquil even in bronchites which are very extensive. The dyspnœa of pyrexia then has the most important rôle. But we know, since the recent experiments of Charles Richet, that this dyspnœa is of central origin, or it may be reflexly provoked. As children and nervous individuals are more susceptible to the influence of excitations than adults and strong men, dyspnœa is always present to a certain extent during their bronchial catarrhs, even when the bronchitis is mild, and the higher the fever, the more short and painful the breathing. In young girls the costo-respiratory type, which is

[1] [Concerning the diagnostic significance of the different râles, it may be proper to add a few remarks. As is well known, the existence of moderate-sized bubbling, muffled râles formerly led at once to the conclusion that fluids were present in the middle-sized bronchi. That this conclusion is inaccurate has been shown by Traube especially. Concerning the crepitant râle of Laennec, it was long ago shown that it also occurs in cases in which, beyond a doubt, no fluid is present in the air passages. As regards the moderate-sized, bubbling, muffled râles, Traube first furnished the evidence of their occurrence even in cases in which there was no fluid in the bronchi. Thus he observed them in cases of commencing pleuritis, and in moderate hydrothorax. From these and other facts Traube justly draws the conclusion that the existence of a fluid in the air passages is by no means a necessary condition for the occurrence of such râles. The similarity between those râles which are produced by liquid in the air passages, and those which certainly occur without the help of a liquid, justify the assumption of a common cause. Traube, therefore, considers as inaccurate Laennec's view that the râles produced by fluid in the air passages are due to the passage of the air in larger or smaller bubbles through the fluid; he attributes them rather to the successive detachment of the viscid bronchial contents, as a whole, from the bronchial wall in the acts of inspiration and expiration. The inspiratory râle is due to the entrance of the air with a certain quickness into the little empty spaces left by the detachment of the bronchial contents. The occurrence of the expiratory râles is to be accounted for in the same manner, according to Traube, only that here the condensation of the air in the pulmonary parenchyma, and not its rarefication, acts as the propulsive power for the penetration of the air between the bronchial wall and the bronchial contents. The râles then which occur without fluid in the air passages are essentially due to the same mechanism.]—Riegel in Ziemssen's Cyclopædia, Vol. IV., Wm. Wood's Ed.

normal, often becomes irregular; in old men dyspnœa is not infrequent. In all the expiratory act is performed with greater difficulty than the inspiratory.

The fever is generally slight and of short duration in bronchial catarrh. As a rule it is higher in the evening than in the morning, the temperature in the early part of the day being normal or nearly so; the pyrexia thus takes on the remittent or intermittent type, lasts several days, or even longer in the case of the infant, and gradually disappears, but never by defervescence as in pneumonia. Rarely it mounts up to 102 ° Fahr., and if it attains this figure there is reason to fear some pulmonary complication.

CHAPTER V.

SEMIOLOGY AND DIAGNOSIS OF INFLUENZAS.[1]

Influenza is benign or grave; whether of much or of little severity, it is characterized by catarrhal irritation of the air passages. Ushering in or accompanying the respiratory phenomena, there is always experienced a series of nervo-muscular or vaso-motor troubles (aching fatigue and fever) and digestive disturbances which are always of febrile origin.

Catarrhs of the Respiratory Passages.

In the midst of the most perfect health and without exposure to a chill as an exciting cause, the disease announces itself by catarrh of the upper or tracheo-bronchial mucous membrane.

Catarrh of the Upper Mucosæ. Coryza.—Influenza commences by coryza, which appears along with the first sensations of discomfort. After frequent repeated attacks of sneezing, there supervenes a discharge, often quite abundant, of a viscid, highly irritating mucus, which by its contact causes a diffuse and painful reddening of the upper lip, which continues swollen and sensitive in proportion to the intensity and duration of the nasal secretion. This secretion, which soon becomes muco-purulent, is the result of a veritable inflammation which by degrees reaches all the neighboring mucous membranes.

Catarrh of the Frontal and Maxillary Sinuses. Cephalalgia and Neuralgia.—It is seldom that the catarrh does not invade the frontal sinuses and interpret itself by a supra-orbital dull and persistent headache, which constitutes one of the most painful symptoms. If the headache is bilateral it is easy to recognize its origin; if it is limited to one side it may be confounded with neuralgia of the supra-orbital or frontal branch of the fifth nerve, an affection which so often follows influenza of the sinuses and which seems to result from a veritable neuritis by propagation of the inflammation to the nerve filaments above mentioned. When the maxillary sinus is invaded, there results a neuritis of the maxillary nerve often mistaken for an essential neuralgia.

Otitis.—Sometimes the catarrh of the nasal fossæ gains the Eustachian tube and the middle ear, producing severe pain, sometimes temporary deafness.

Conjunctivitis.—The conjunctivæ are often primarily invaded; the conjunctivitis seems in this case to fix and to exhaust the disease, but generally the irritation of the conjunctivæ is only the effect of the ascending march of the nasal catarrh. In both cases the mucous membrane is red and injected; there is lachrymation with or without painful photophobia.

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[1] This chapter was written with the collaboration of my clinical chief, Dr. A. Matthieu.

Irritation of the Pharyngeal, Laryngeal, Tracheo-bronchial Mucous Membranes. Erythematous Angina.—Instead of invading the anterior nares, sinuses and conjunctivæ, the irritation gains the posterior pharynx, and produces an erythematous angina marked by a lively redness, by most annoying snuffles, by a nasal and guttural voice, and impeded breathing.

Laryngitis.—From the throat the catarrh generally extends to the larynx, thence to the trachea and large bronchi, but this progressively descending march is not constant; one or more of the mucous tracts may be spared by the inflammation, which, as it were, overleaps them or makes no appreciable stay there. The laryngitis manifests itself by hoarseness of the voice, which becomes extinct at times, to take on presently a shrill character, the words or sentences being occasionally broken by a too acute (falsetto) rise, or by an exaggerated amount of tone imparted to the utterances; these vocal phenomena are due to inflammatory constriction of the chordæ vocales, and to the interposition of a plug of mucus. The infrequent, always distressing cough, sometimes ends in expelling a certain amount of mucosities, which are thick, discrete and small, like pearls; in fine the larynx may be painful to pressure over the thyroid cartilage.

Tracheitis.—When the trachea is invaded, which is far from being the rule, the patient experiences a retro-sternal pain, a sensation of burning during the passage of the respired air, a paroxysmal, painful, dry cough without expectoration, at least in the onset, and without any râles audible to auscultation.

Bronchitis of the Large and Medium Sized Bronchi.—Often the trachea and larnyx remain exempt, and the violence of the catarrhal affection falls upon the thoracic organs. The bronchitis of the larger tubes is in fact as constant as the coryza; it manifests itself by the habitual signs of bronchial catarrh, namely by a certain amount of disseminated râles, at first dry, sibilant or snoring, then as fast as the catarrhal secretion becomes loose and fluid, we have sub-mucous sub-crepitant râles, often in clusters, oftener few and scattering, and extending to the upper or middle lobes of the lungs; always bilateral, which constitutes one of the principal characteristics of the bronchitis of influenza.

Neuro-muscular and Thermic Troubles.

Along with and sometimes even before the mucous catarrh, there is manifested on the part of the muscular and peripheral nervous system a series of profound disturbances which may be comprehended under the name of painful neuro-muscular fatigue; we refer to the physical prostration, the aching in the limbs and back, the cephalalgia. These phenomena are generally the indices of the invasion of the organism by microphytic diseases, and this again affiliates influenza to maladies of this kind.

Moreover there supervenes a febrile movement of ascensional type, which equally belongs to the development of the infectious agent.

Prostration of the Muscular Forces.—The patient experiences from the commencement, a general feebleness, a prostration which is out of proportion to the irritation of the mucous membranes, and to the febrile movement; it is a sort of muscular exhaustion which renders him incapable of all effort, of all movement. This physical depression entails not only inaptitude for intellectual work but also a profound mental depression, and often, in the case of females, frequent attacks of fainting.

The countenance bears the impress of this general depression; the features are sunken, the muscles of the face participating in this lowering of nervo-muscular tone. At the same time it is worthy of note that the expression of the countenance in no respect resembles the typhoid hebetude, the stupid listless aspect which is essentially of psychical order and which marks the onset of typhoid fever.

Painful Aching of the Muscles.—The muscular incapacity is accompanied by keenly painful sensations which seem to the patient to have their source in a general contusion; these uncomfortable feelings localize themselves in the limbs and in the dorso-lumbar, or dorso-costal region.

Peri-articular Pains.—In the extremities these occupy especially the neighborhood of the tendinous attachments of the muscles and their fibrous expansions; sometimes the joints themselves seem involved, and so severe is the pain as to lead the patient to fancy himself attacked by acute articular rheumatism; but if you analyze with some attention these localizations, you can easily assure yourself of their superficial peri-articular seat, *i.e.* their location in the fibrous tissues of the vicinity of the joint, while the ligaments themselves and the synovial membrane remain quite exempt. In the course of the muscles, painful cramps have been noted; these seem to indicate a sort of arterial ischæmia of the muscles like that which is observed in the fatigue which succeeds a forced march.

Rachialgia.—The muscles of the dorsal and lumbar regions often participate in the painful muscular fatigue which the entire system undergoes. Rachialgia and lumbago are the habitual accompaniment of the peri-articular pains.

Pleurodynia.—It is the same with respect to the pleurodynia, or in other words, the myopathy of the pectoral or intercostal muscles, which coincides with the other nervo-muscular sensations, and marks still oftener the invasion of the subjacent organs, namely the bronchi or the lungs.

Cephalalgia.—The headache in influenza is a frequent and initial symptom. It presents itself in divers forms and under divers influences. Sometimes this cephalalgia consists in general epicranial pains which are of muscular order, and which occupy the teguments with the subjacent muscular layers; in this case there are violent twinges either arising without any known cause, or determined by pressure over the tract of the nerves; at other times we have to do with veritable neuralgias of the frontal and supra orbital and maxillary branches of the fifth; these neuralgias are generally unilateral and consequently easy to distinguish from the epicranial pains. Finally, under the influence of the catarrhal inflammation of the frontal and maxillary sinuses, veritable neuroses may be produced by propagation and persist for a variable time; this is a frequent consequence of the coryza of influenza.

Fever.—Fever is almost always present, with variable intensity and a type almost uniform. Rarely it declares itself suddenly; generally it begins gradually by a temperature of 38° to 38.5° C. (100°–102° Fahr.) in the evening, presents a considerable remission in the morning, falls to 37.5° or 38°C. (99° to 100° Fahr.), and continues in this way, with oscillations, for three or four days, to mount up again in the evening to 38.5° or 39° (102°–102½° Fahr.) and even more. This is exactly the type of measles, between which and influenza we shall have to establish the differential diagnosis, sometimes quite a difficult matter. It is, moreover, the common type of typhoid fever, but with this difference, that in influenza the morning re-

mission is complete and brings with it the temporary cessation of the painful aching and the malaise.

Digestive and Secretory Troubles.

Historians of epidemics, guided doubtless by the impression that the particular epidemic which they are describing has certain peculiarities which differentiate it from all other epidemics, are prone to insist on anomalous aspects which are seldom of sufficient importance to constitute distinct types, and to multiply groups beyond necessity; hence it is that writers have reckoned three different varieties of influenza; namely the mucous form, which really comprehends the disease in its entirety; then the adynamic form, which is but the resultant of the phenomena of muscular fatigue, and finally a gastric or gastro-intestinal influenza, which I regard only as the effect of the fever and the muscular prostration.

Gastric Phenomena. Catarrh of the Buccal and Gastric Mucous Membrane.—From the very first the patient complains of loss of appetite, nausea, more rarely of vomiting, which seems to depend on some degree of catarrh of the stomach. The lingual mucous membrane is the seat of a muco-epithelial yellowish or white exudation of considerable consistency, streaked with red along its borders; this state indicates a local secretory trouble such as manifests itself at the commencement of all fevers and infectious diseases, and suffices to explain the anorexia.

Ectasy of the Stomach.—In several patients affected during the last epidemic, my clinical chief Matthieu noted a painful dilatation of the stomach which disappeared with convalescence; this is moreover an adynamic phenomenon of the muscular layer of the walls of the stomach: we see here, then, the influence of the adynamic and secretory troubles.

State of the Intestines and Liver.—The intestinal functions undergo perturbations of the same kind; sometimes it is constipation, which exists by reason of the lowered tonicity of the muscular system; sometimes it is an abundant mucous, almost dysenteric diarrhœa, from predominance of the catarrh.

Lastly, it is not rare to observe certain troubles in the biliary secretion, which may possibly be the effect of propagation of the intestinal irritation to the bile ducts. To sum up: We have here simply the repetition of what takes place in the respiratory passages and the muscular system, but these gastro-intestinal phenomena never exist alone and should not be arrayed into a separate category.

Semeiology of the Grave Forms.

Influenza does not always confine itself to these superficial irritations of the respiratory mucous membranes; it often takes on the form of deep or parenchymatous inflammation of the respiratory organs; sometimes also the infectant character. Let us note especially the grave lesions of the bronchi, the lungs and the pleura.

Grave Broncho-pulmonary Influenza. Bronchiolitis.—In infants the bronchitis may even gain the pulmonary bronchi, take on the form of bronchiolitis, and manifest itself by suffocative catarrh, with few or no auscultatory signs.

This last form is often noticed in emphysematous patients and in old people; in the latter for the reason that the senile lung is naturally

emphysematous. The continued oppression, the subtympanitic sonorousness, the respiratory sibilance (of which we shall speak shortly), leave no doubt as to the coexistence with the influenza of pulmonary emphysema.

In the adult it is seldom that the inflammation overpasses the ordinary limits of tracheo-bronchitis.

Broncho-pneumonia.—But it may happen even in the adult that the influenza may attack the pulmonary vesicles and constitute a veritable broncho-alveolitis or broncho-pneumonia. If the inflammatory foci are multiple and contiguous, you will have all the signs of lobar broncho-pneumonia (dullness, tubular breathing, subcrepitant râles, then bloody expectoration, etc.), with all the signs more or less complete of the initial bronchitis, which has, so to speak, introduced the infectious principle into the terminal bronchi.

Migratory Pneumonia.—During the epidemics of influenza it is a common thing to encounter pneumonic cases (See Part II.) which along with fever and the characteristic expectoration, are marked by dullness in a third or half of one lung, with blowing breathing, bronchophony, and, above the boundaries of the dull zone, fine subcrepitant râles. Then from two to four days afterwards, the seat of the dullness and the souffle is found higher up or more in front, and at the primitive point of invasion you hear a few large returning crepitant râles. Then the pneumonia shifts place again, and attains the posterior scapular region or even the opposite side: this is the type of migrating pneumonia, which undergoes evolution more slowly and less abruptly than common infectious pneumonia, with which one might easily confound it, if there were not present the prodromic symptoms of irritation of the upper mucous passages.

Pleuro-pneumonia.—Pleuro-pneumonia, although quite rare, may be said occasionally to exist; it is distinguished especially by the intensity of the pleurodynia with hyperalgesia, which propagates itself from the ribs to the spine and sternum, and by all the concomitant signs of broncho pleuro-pneumonia. The effusion often gains the upper part of the thorax, where it spreads laterally. It is easy to follow its progress by the extension of the dullness and the line of ægophony.

After several days of oscillation, the pleuro-pneumonic focus becomes circumscribed, shrinks, then the liquid is absorbed, the souffle and the ægophony disappear, and there remains of the vanished lesion nothing but a *bruit de frottement* more or less persistent.

Grave Infectant Influenza.—In some cases the gravity of the influenza reveals itself by infectant lesions of the lymphatic glands, the tonsils, the spleen, or the secretory organs, and the kidneys.

Angina.—The grippal[1] Angina may take on a grave and preponderant character; generally it is quite superficial and occupies, always as a sequel of the coryza, the mucous membrane of the velum pendulum, the uvula and the half arches; these have a lively red, often violaceous color, are sometimes mottled with scattering reddish dark spots as in measles; very rarely there are pultaceous deposits as in scarlatina.

Amygdalitis.—But the influenza does not always limit itself to these regions, it may invade the tonsils, which become red, swollen, livid; the openings of the mucous follicles are patulous, distended, and give exit to a tuft-like discharge of desquamated epithelial products which accumulate

[1] *Grippal*, i.e., pertaining to influenza (grippe, Fr.).

in their cavity. There results from this an aspect which is quite peculiar. Generally one of the tonsils is much more swollen than the other and threatens to suppurate. The part projecting in front of the anterior half arch is tense, tumefied and of shiny appearance. By palpation with the extremity of the index finger, you perceive a marked elastic resistance and increased density of the tonsil. When pus forms in the interior, you notice a fluctuation more or less well defined, more or less superficial, according to the situation and extent of the purulent focus; in these cases the jaws are almost set, the voice nasal, and the sub-maxillary region swollen. At the same time there is an intense fever, which mounts as high as 40°C. (104° Fahr.) and is accompanied with sub-delirium. Bouchard believes that in these cases there is resorption of pyretogenous products.

State of the Spleen and Kidneys.—One fact which gives support to the supposition that influenza is of parasitic nature, is that in grave cases, as we have ourselves observed many times, the spleen is augmented in volume and is sometimes painful. This hypertrophy was noticed and pointed out in 1847 by Peacock, who believed it typical of intestinal influenzas; a slight critical examination, however, suffices to show that Peacock mistook cases of typhoid fever for influenza at a time when French researches on dothinenteritis had not yet penetrated England. The fact of the splenic alteration is interesting from a doctrinal standpoint, being suggestive of the specificity of the disease, which always seems so simple and so benign.

The state of the kidneys, or rather of the urine, is equally significant. In the grave forms, especially in the amygdalitis, or in the grippal pneumonia, it is not rare to find albuminuria, which points to a condition of the kidney analogous to that of the spleen.

Cerebro-meningeal Lesions.—Raymond has recently related the history of a patient who, as he believed, succumbed to the consequences of suppurative meningitis, resulting from cerebral influenza.

Diagnosis of Influenza.

It is quite a common thing to meet difficulties of diagnosis between influenza and typhoid fever and between the former disease and miliary tuberculosis.

Typhoid Fever.—The difficulty with respect to typhoid fever may be removed by attentively taking into account the march of the pyrexia, characteristic of this disease, and the diazotic reaction of the urine.

In the grave cases of influenza, especially where there is some pulmonary complication, the febrile type peculiar to this affection may be temporarily interrupted or disturbed. It often seems as though a tracing of pneumonia were intercalated into a tracing of influenza. At the beginning and at the end, the morning remissions are accentuated; during the period which corresponds to the pneumonia the thermometric elevation, morning and evening, continues from 39° to 40° C. (102° to 104° Fahr.).

When the pulmonary manifestations consist in a generalized persistent bronchitis which resembles by its symptoms capillary bronchitis, or even acute tuberculosis, the quotidian type with matutinal remissions is usually witnessed. This character, joined to that of the known existence of an actual epidemic of influenza, may enable you to make a diagnosis which other circumstances may render problematical and difficult.

Diazotic Urine.[1]—We have already pointed out this precious means of diagnosis (See "Bacillary Phthisis," p. 327,) when it is a question of determining acute tuberculosis by the urine. Typhoid fever is equally amenable to the same test. During the last epidemic of influenza, we were often unable to arrive at a diagnosis till we had resorted to the diazotic test of the urine; a peculiar color reaction being thereby obtained, which is constant in typhoid fever from the first three days, but which is always wanting in influenza.

Tuberculosis.—It is not always easy to diagnosticate influenza from acute tuberculosis, although the diazotic reaction, as before intimated, is a precious test. In obscure cases the examination of the sputum clears up all doubt. As soon as there is a consistent expectoration, composed of muco-pus, you can be sure if you find no bacilli there, that it is a case of influenza. The presence of the microphyte in the sputa gives certainty as to the existence of tuberculosis. In our first volume are given several examples of tuberculosis mistaken for influenza, and diagnosticated by the presence of the parasites.

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[1] The diazotic compounds, which result from the action of nitrous acid on the amides of the aromatic series, are distinguished by a special coloring property. Thus from nitrite of diabenzol and of aniline, there is formed during the evolution of nitrous acid, an amido-azo-benzol, a yellow coloring matter. Ehrlich has turned to profit this coloring property, to make his diazo compounds serve as means to detect principles in the urine before undetermined. He employs the sulphanilic acid of commerce, of which a certain indeterminate quantity is added to a pint of water further acidulated with 50 c.c. of nitrous acid; a few grains of nitrate of soda in a watery solution are now added. To obtain the reaction, you dose a cubic centimetre of urine with an equal quantity of the reagent and saturate with ammonia; let the whole rest for twenty-four hours. Normal urine remains indifferent; it is the same with all non-febrile urine, except in phthisis: in most samples of urine of febrile diseases a scarlet or orange-red tint is observed. Somewhat later the red coloring matter is dissolved and transformed into an insoluble emerald green, and thus colors the superficial stratum of the sediment. In typhoid fever this reaction is seldom wanting; it appears on the evening of the second day. The intensity of the coloration generally corresponds to the intensity of the malady. The same singular reaction also characterizes the urine of most cases of phthisis. Professor Sée, out of forty cases, did not find it to fail in a single instance. The two colors—first red, then green, are almost constant, but in a few instances only the green color appeared; this Ehrlich considers only a sequel of the first hue. Escherich, out of twenty patients in phthisis, found the urine in eighteen colored a lively red by the sulphanilic acid.

CHAPTER VI.

CAPILLARY BRONCHITIS

Definition—Etiology.

Anatomical Considerations. — Acute bronchitis may invade simultaneously the bronchi of more than a millimetre in diameter, and which are provided with a muscular coat and glands, the intra-lobular bronchi and the alveolar or terminal bronchi.

In the FINE bronchi the epithelial covering is constituted by round cells coming from the capillaries by diapedesis and from the deep strata of the mucosa; these may compress the other cells and render them polyhedral; always they tend to replace the cylindrical (columnar) cells of the surface, possessing like them vibratile cilia. One result of inflammation, at least in experimental bronchitis—is not the rapid accumulation of cells on the surface, as one would expect, but the destruction and desquamation of the epithelium, which is replaced by a layer of lymph cells, the mingling of which with the liquid exudation of the blood-vessels forms the muco-purulent secretion which lines the bronchus; the epithelial cells which remain intact preserve their basement membrane. But amid these morbid changes, notwithstanding all the lesions of the superficial epithelium, the surface of the mucous membrane is never ulcerated. It seems uneven, irregular,—deprived of its usual polish—but there is no loss of substance appreciable to the naked eye. These are then the important facts: diapedesis of lymph cells, fall of epithelium, but no ulceration. At a more advanced stage the connective tissue of the mucosa and the intra-muscular cellular tissue are also infiltrated with lymphoid cells and are often thickened to such an extent as to impede the action of the unstriped muscles; this constitutes the principal cause of bronchial dilatations.

Intra-lobular Bronchi. — The bronchi which penetrate the lobules have, in their physiological condition, but one muscular tunic, which is very incomplete, and relatively few vibratile cells, disseminated in the connective tissue which forms the septum of the neighboring alveoli.

In capillary or intra-lobular bronchitis the layer of cylindrical cells remains in place; the lymph cells fill the bronchial cavity and infiltrate the cellular tissue of the bronchial wall as well in its sub-epithelial stratum as in its external part which is in relation with the pulmonary alveoli and the arteriole itself, so that the air tube is surrounded by a circular zone formed of connective tissue infiltrated with lymphoid cells.

Alveolar or Terminal Bronchi. — From the intra-lobular bronchus (central bronchus of the lobule), issue the terminal bronchi which are lost in the pulmonary alveoli. It results from this intimate connection of the intra-lobular bronchus with the terminal bronchi and the pulmonary alveoli, that the inflammation of an intra-lobular bronchus involves almost always the entire pulmonary lobule; thus constituting a

lobular broncho-pneumonia, that is to say a pneumonia of bronchial origin which must be kept separate and distinct from lobar pneumonia—always a primitive disease, always parasitic, and quite independent of all catarrhal inflammation of the air tubes. But from a merely anatomical point of view the distinction is far from being absolute, for the character assigned to lobar pneumonia, to wit, the intra-alveolar fibrinous exudation, may be present in broncho-pneumonia. The real difference between these two kinds of inflammation, so nearly alike with respect to the alveolar lesion and its nature, consists in the mechanism and etiology, which, as before said, is not the same in the two affections.

Clinical Definition.—The conclusion to which physiological and pathological anatomy and histology lead, justifies fully the diverging views which have been held since the time of Laennec respecting the independence of bronchiolitis or its fusion with lobular pneumonia. Laennec gave to suffocative catarrh, which was known to clinical medicine, an anatomical existence, in assigning for its basis capillary bronchitis, and while this capillary bronchitis figured, by reason of the writings of Fauvel, Hardy, and Behier, in the general inflammations of the bronchi, even when it extended to the alveoli, it disappeared from the classifications of nosologists after and in consequence of the labors of Legendre and Bailly, who gave especial prominence to atelectasis, that is to say, the collapse of the pulmonary lobules obliterated by the mucus of the inflamed bronchioles; it was treated slightingly by Rilliet and Barthez, who entitle their chapter: *Capillary bronchitis or broncho-pneumonia;* it was relegated to an inferior place by Roger and Damaschino in their excellent study of broncho-pneumonia; vehemently attacked by Picot and Espine, who call it suffocative broncho-pneumonia, in fine by Charcot and Balzer, who in their interesting researches on the lesions of broncho-pneumonia scarcely make mention of the real point of departure. But it is precisely in taking pathological anatomy for their exclusive basis that authorities have arrived at complete negation of the disease. In fact the alternatives have been well stated by Blachez and Cadet de Gassicourt:—"either capillary bronchitis resulting from a general catarrh, limits itself to a small number of bronchioles and ends by comporting itself like a general bronchitis which rapidly gets well; or else in obliterating the bronchioles it kills by asphyxia, after having barely lasted several days;"—(here Cadet de Gassicourt cites the case of a patient where there was not a trace of lobular pneumonia found at the autopsy); —"or finally it advances towards the alveoli and determines a lobular pneumonia;" this is what generally happens, and this is why the autopsies have almost always proved the existence of a broncho pulmonary inflammation. It is no less true that we are justified in recognizing three types of capillary bronchitis which present themselves with clinical characters often sufficiently precise to warrant the diagnosis:—diffuse or fine catarrhal bronchitis; bronchiolitis, or inflammation of the intra-lobular branches; broncho-alveolitis, or inflammation of the alveolar bronchi; these three types constitute the group of capillary bronchites.

Pathogeny.

Effects of Bronchitis Obliterans.—Under the collective name of capillary bronchitis we shall designate henceforth diffuse bronchitis of the fine bronchi, lobular bronchiolitis and alveolitis. But in all this morbid group we find two kinds of effects sure to follow, and quite dis-

tinct: these are (a) such sequences as attend obliteration of the bronchioles, and (b) those which result from the inflammation. The mechanical element (*i.e.* the obstruction) predominates in inflammation of the bronchioles, which by reason of their small size are predisposed to become obstructed: this is especially the case in young children. The inflammatory element constitutes the common basis of all the capillary forms of bronchitis, but especially of the broncho-alveolar catarrhs. Among the effects of bronchial obstruction we reckon; 1, asphyxia; 2, pulmonary atelectasis; 3, emphysema. The inflammation dominates all the other lesions,—to wit the muco-purulent secretion, and the congestion to which has been assigned a preponderant rôle, although according to the admission of its most confident asserters (Damaschino and Cadet de Gassicourt) it is not found after death and does not always exist even during life.

Asphyxia.—The dangers attending capillary bronchitis are easily foreseen. They result from diminution of the field of hæmatosis by reason of the extension of the catarrhal inflammation; the bronchioles are swollen, and either do not suffer the air to pass, or they allow it to pass with great difficulty; finally a certain number of the lobules are shut off from the exercise of their function because the air does not enter them; the exchange of gases fails properly to take place, and carbonic acid accumulates to dangerous excess in the pulmonary and systemic vessels, whence result attacks of suffocatiou—a veritable asphyxia.

Atelectasis.—One of the most remarkable and most constant effects of the obstruction of a bronchiole by the concrete mucus of the catarrhal inflammation, is the collapse of the corresponding lobule. This condition was admirably described forty years ago by Legendre and Bailly, under the name of *fœtal state* of the lung (*état fœtal*), and at an earlier date by Jorg under the name of atelectasis.

The collapsed lobule, deprived of air, becomes flaccid, non-crepitant, dense as muscular tissue, of a bluish dark color, easily distensible by insufflation of air, and consequently capable of regaining its primitive condition. In these lobules, the sunken and flattened alveoli can be distinguished with their dilated blood-vessels; they contain a few red globules and granular cells, but no true inflammatory products.

Mechanism of the Atelectasis.—Everybody admits that atelectasis is due to obstruction of a bronchus by the muco-purulent exudation, Gardner, however, claims that the bronchial plug which stops the entry of air into the vesicles is unable to prevent the expulsion of the air, by reason of the predominance of the expiratory forces. As a result of this mechanism, the air of the alveoli is gradually forced out by expiration till the vesicles become empty and their walls sink together. Other observers think, with Virchow, Fuchs, Ziemssen, Grancher, etc., that the alveolus, its supply of air being cut off, instead of expelling its residual air, allows it to be removed by absorption. It matters little which explanation you take; the bronchial obstruction exists, but it is well to bear in mind that this is not the only or the principal morbid phenomenon concerned in the production of broncho-pneumonia.

Emphysema.—A third mechanical effect of the obstruction of the bronchi is vesicular emphysema, which is either general, or limited to the front and upper regions of the lungs. The lung resembles in these portions, an inflated bladder, and does not sink in when the chest is opened. Parts of the lung have become collapsed and hepatized, but neighboring (compensating) lobules, by dint of extra work and forced

dilatation have lost their elasticity; this has been called vicarious emphysema, but the vicariating lobules are unable to assist in the function of hæmatosis. Around dense, shrivelled nodules, then, of splenized lung, you find emphysematous projections which fill the vacancy caused by collapse of the lobules. (Gardner.)

In fine the emphysema may become sub-pleural as well as inter-lobular.

As the emphysema is always an effect, it does not belong to us here to consider it except in its relation to diagnosis, in respect to which it is always an important factor.

Divers Modes of Development of the Bronchites and Alveolites.

It is demonstrated that lobular pneumonia is in close relation with bronchial catarrh, which is the constant accompaniment as it is the precursor of the pulmonary inaflammation; but how does the bronchitis lead to the pneumonia?

Inflammation by Reason of Obliteration.—An opinion often put forth and oftener contested, regards the facts in this light: the bronchitis leads to obstruction by the mucous plug, this to lobular atelectasis by absorption of the air, and the atelectasis gives rise to inflammation of the lobules, which is characterized by extravasation of serum and diapedesis of the white globules, also by tumefaction and fatty degeneration of the alveolar epithelium, and proliferation of the cellular elements. This is the view held by Ziemssen and Bartels, who think that the bronchial exudation extends as far as the alveolus whose inflammation it determines.

Lobular Inflammation by Propagation.—According to the view of the French physicians, Charcot, Balzer, and Cadet, the inflammation of the bronchi progresses little by little towards the alveoli; it is at the side of, and not in the collapsed lobules that you find what is called splenization, *i.e.*, a congestive inflammation. Close by the shrunken flattened lobules you find others which only show traces of congestion, and still others which are the seat of a true inflammation of congestive type. The inflamed lobule is distinguished from the fœtal state of the lung (*état fœtal*) by its resistance to insufflation. Bouchut, in 1845, in vain protested against this means of diagnosis; he saw clearly that all the lobules, even those that were hepatized, might become again permeable to air. But it needed a new and more thorough method of examination, in the light of modern histology, to show that the salient lobules differ from the collapsed lobules:—the constituent elements, the lesions which characterize inflammation, the induration of tissue, the thickening of the inter-lobular septa, and even the alteration of the alveolar epithelium, all this is found in those hard projecting nodules which are seated generally in the posterior part, or in the tongue-like processes and borders of the lungs. Bartels has even noticed the existence of lobular inflammations without obstruction of the bronchi, and he has imagined a reflex spasmodic condition of the bronchi, as a sufficient cause of obliteration of the air tubes. Clinical medicine and experimental physiology agree in establishing the perfect independence of lobular inflammation and atelectasis.

Experimental Lobular Inflammation.—Frey, repeating the remarkable experiments of Traube pertaining to the production of artificial pneumonia by section of the pneumogastric, has had ocular evidence

that this pneumonia does not result from collapse of the lung, but from the introduction of buccal mucosities and fragments of food into the air passages; the condition being that these substances shall reach the bronchi, and what is better, that they shall be aspirated into the pulmonary alveoli, as easily happens during inspiration in a fit of dyspnœa. On the other hand, the neuro-paralytic pneumonia does not develop when by any means the entrance of these matters into the air passages is prevented; one may even, without practicing the section of the vagi, obtain artificial pneumonia by the sole fact of production of these alimentary emboli.

The Inflammation is either by Continuity and Contiguity, or it is Parasitic.—It is evident, then, that besides the obliteration of the bronchi there is needed in order to provoke pulmonary inflammation, an irritant agent. It is also demonstrated that bronchial inflammation may propagate itself to the alveolar tissue; one proof of this is that the pneumonic process may spread to the alveoli which surround the bronchial walls and constitute a peri-bronchitis. It is a matter of observation, too, that the first alveolar groups which undergo inflammation are in direct continuity with the inflamed bronchi.

But to this cause there is ordinarily joined another which dominates the entire pathological series; namely the infectious agent. The greater part of the broncho-pneumonias succeed the specific catarrhs, such as measles, whooping cough, influenza, diphtheria, in all of which events there exist infiltrations of bacteria in the alveoli, in the lymph spaces, and lobular connective tissue.

It results from this: 1, that lobular pneumonia has bronchial catarrh for its starting point; 2, that generally obstruction of the bronchioles is an intermediate stage in the development of the disease; 3, that the inflammatory process may advance by continuity and contiguity to the alveoli; 4, but it is above all necessary to take into account as chief causal factors, irritants of every kind, and especially the infectious agents which may directly engender the alveolar phlegmasia. This seems to me the broadest and most scientific way to explain and to treat these capillary bronchites, these broncho-pneumonias which for half a century have been subjects of contention, because assigned to the too exclusive domain of pathological anatomy.

Individual Factors of Causation.

Capillary bronchitis is almost always a bronchitis which has pursued its march from the large bronchi to the bronchioles and alveolar bronchi. I insist, with Cadet de Gassicourt, in maintaining that this is not a secondary affection of the smaller bronchi, but an extension, in all its intensity, of a disease originating in the larger tubes. It may even happen that the capillary disease may be primary, manifesting itself first.

However this may be, capillary bronchitis has its point of departure in catarrhal bronchitis of some part of the bronchial tree, whether the catarrh be the result of cold, or, what is more frequently the case, a specific cause, such as influenza, measles or diphtheria; it may gain the smaller bronchi and determine bronchiolitis or broncho-pneumonia, which derives a special character of gravity from the parasitic origin of those diseases.

Influence of Childhood. — The frequency of the capillary bronchites at the different ages is, then, regulated, first and chiefly by the frequency of the parasitic diseases, with inevitable

catarrhal manifestations, such as pertussis and measles. These diseases are especially prevalent among children who are the most exposed to the contagion, namely those who are congregated in groups (children of infant asylums, boarding schools, work-shops, etc. As, moreover, these two diseases are generally non-recurrent, it follows that capillary bronchitis shares their invasions as well as their immunities; thus it is that measles is oftenest observed between the ages of two years and six, and this is why the specific capillary catarrh predominates at this time of life.

Ages Subject to the Influenza-bronchites.—With regard to influenza there is no difference as respects age, all the inhabitants of a country invaded by this epidemic may be smitten indiscriminately, but adults resist the disease better than young children and old people. Physiology and anatomy explain perfectly the danger of grippal bronchitis in children: the ultimate bronchi have a calibre which diminishes rapidly and is not proportioned to the exigencies of hæmatosis. The least exudation is sufficient to obliterate the bronchioles and determine the formation of alveolar bronchitis.

In old age another cause intervenes; namely, a tendency to blood stasis resulting from troubles of the circulation; *i.e.*, senile alterations of the heart and systemic vessels. These passive hyperæmias more readily provoke a bronchial hyper-secretion than the active congestions, and when the exudations take place they are expectorated with difficulty, they accumulate and determine the obstruction of the smaller bronchi.

Prevalence of Epidemics of Measles and Influenza among Young Soldiers.—Since 1841 there have been several terrible epidemics of capillary bronchitis among young soldiers, attended with a mortality of from two to three fifths of those attacked. The physicians of Nantes observed the first epidemic during a season of dry cold weather while measles and scarlatina were raging. At St. Omer, at Lyons, at Paris, at Boulogne, the same malady also prevailed among the recruits, sparing the old soldiers and the officers, and spread to considerable extent by contagion. It was a kind of suffocative catarrh with enormous purulent secretion of the bronchi, manifesting itself during life by asphyxiating phenomena, and general râles; after death by simple bronchitis or broncho-pneumonia, so well described by Colin.

Ages Attacked by Common Bronchial Catarrh.—Abstraction being made of specific causes, there remains the ordinary cause of cold, which is unquestionable; cold may in fact directly provoke broncho-pneumonia. It begins by determining a simple catarrh, which in certain states of mal-conformation of the thorax or of general debility, propagates itself to the terminal bronchi. Nurslings are, they say, less liable than older children to take cold from exposure outdoors; this is possible, but how is it within doors? Do we not see young children badly cared for, bathed without any precaution, scantily clothed or sweltering in thick underclothing which is impregnated with foul secretions from whose presence chilling readily ensues? Broncho-pneumonia does not select its conditions of chilling, and a severe cold, however acquired, may easily provoke a catarrh, which beginning in bronchitis ends in alveolitis.

Older children, those for instance between two and five years of age, furnish a smaller contingent of victims to the capillary disease, although they take colds more readily and oftener. The disease attacks the rickety and hump-backed because the deformed thorax easily allows the products of muco-purulent secretion to accumulate. Capillary bronchitis affects

also children debilitated by bad hygiene or by chronic diseases which have enfeebled the respiratory muscular system and especially the muscular mechanism of expiration, so that the young patients are unable to cough to good effect and bring up the accumulated mucosities; their breathing also is not energetic enough to prevent the stagnation of air in the alveoli. In any case the strong healthy child with good muscles, of good physiological conformation, does not readily take capillary bronchitis. This disease, according to Roger, is primitive in a third of the cases, which doubtless bear relation to local and general conditions, which we have studied thus minutely by reason of their extreme importance from the point of view of preservation and of treatment.

CHAPTER VII.

SEMEIOLOGY OF THE CAPILLARY BRONCHITES.

The semeiology cannot be well understood or elucidated unless three types of capillary bronchitis are admitted, to wit: 1, bronchitis localized in the fine bronchi; 2, bronchiolitis of the lobular branches, either isolated or grouped in mass so as to form pseudo-lobar pneumonia; 3, the alveolitis which often succeeds the preceding (*i.e.*, the bronchiolitis) and represents especially the inflammatory form, while the bronchiolitis manifests itself by mechanical phenomena. The first is a diffuse capillary bronchitis. The second is a bronchitis with lobular foci, the third a bronchitis with lobular foci inflamed. This distinction is certainly a difficult one, but you will now and then meet with cases that justify it.

Capillary Bronchitis by Extension.

A general bronchitis which has spread to the capillary bronchi, or a bronchitis which is capillary from the onset, manifests itself always, 1, by dyspnœa, which becomes finally asphyxiating, and is accompanied by cerebral and circulatory troubles resulting from the asphyxia; 2, by general diffuse mucous râles, or even fine bubbling râles, without any modification of the percussion sound; 3, by a variable cough with difficult expectoration; 4, generally by febrile accompaniments.

Dyspnœa. Suffocative Catarrh.—The respiration is always accelerated, almost always impeded; the dyspnœa, which is apparent in both parts of the respiratory act, develops slowly and only attains its maximum at the end of several days, to be then subject to numerous oscillations. The difficulties of respiration naturally increase with the extension and degree of inflammation of the small bronchi. The more impermeable these are by reason of swelling of the mucosa, and especially by reason of accumulation of mucus, the more pronounced is the dyspnœa; when the secretion is expelled by the cough, and the bronchial tube is cleared so as to give passage to the air, the respiration becomes calm, to become embarrassed again when the mucosities collect anew. When the obstruction is considerable and invades a large number of the capillary tubes without even reaching the lobular branches, the dyspnœa may even amount to suffocation. In these cases the gaseous exchanges in the lungs are more and more limited, and there results the series of phenomena which characterize asphyxia, and we have ushered in suffocative catarrh, asphyxiating dyspnœa, so different from febrile dyspnœa.

Cyanosis.—The cyanosis manifests itself in the extremities and in the countenance: the lips, the ears, the nose take on a violaceous leaden hue; the circulation is enfeebled, the pulse becomes small and accelerated.

Type of Asphyxiating Respiration.—The respiration demands now the co-operation of all the auxiliary muscles, the scaleni, sterno-cleido

mastoidei; each respiration is short, interrupted, superficial, and the thorax does not undergo perceptible expansion. The larynx sinks at each inspiration, carrying with it the jugular furrow, and the sternum inferiorly and the lower ribs are retracted. The dyspnœa is aggravated by expiration as well as by inspiration, and the expiratory period is prolonged, especially in the infant.

Form of the Thorax in Dyspnœa. Attitudes.—Quite an important sign of the disturbance of gaseous exchanges, *i.e.*, of the asphyxiating dyspnœa, is the inspiratory rĕtraction of the thorax. In the adult this retraction is limited to the intercostal spaces, particularly the inferior intercostal spaces, and is noticed sometimes even during health at the commencement of inspiration. In the infant the thorax being flexible, the retraction affects the costal cartilages and the ribs as well as the xiphoid appendix, the epigastrium, and the two hypochondria, which undergo considerable depression. The explanation of this is that the entrance of air into the alveoli is interrupted, so that during the inspiratory dilatation of the thorax the lobules, cut off from their supply of air, can take no part in the dilatation, and the flexible and superficial portions of the thorax are under pressure from the outside atmosphere, and forced inwards. You see under these circumstances in the infant, a sort of antagonism between the superior and inferior segments of the chest. While the inferior portions undergo deep depression at each inspiration, the thorax superiorly seems protuberant in front, and almost immovable. It is easy to see that the upper lobes are then in a state of subacute emphysematous distention, and of fixed inspiratory dilatation; this condition results from the fact that during inspiration some air can without doubt reach the alveoli through the obstructed bronchi, but during expiration, even though this be aided by the auxiliary muscles of the abdomen, the alveoli empty themselves incompletely, and remain distended with air. From this results a certain degree of alveolar emphysema, which also opposes the inspiratory aspiration of the air.

Asphyxiating Dyspnœa.—During the respiratory efforts, the adult patient adopts passive or forced attitudes; he is obliged to breathe in a sitting posture, and is orthopnœic; in the infant, too, the want of air augments from dorsal position, and instinctively the little sufferer demands to be carried in the arms of its mother or nurse. All the activity of the auxiliary muscles of respiration, as well as these instinctive attitudes, have for their end to facilitate the respiratory act.

Consequences. Blood Stases.—The circulation in its turn participates in the trouble of respiration; the afflux of venous blood to the heart is interrupted; the aspiration of the blood from the venæ cavæ is incompletely performed. You recognize these stases not only by the cyanosis of the teguments, which is the necessary result of the imperfect decarbonization of the blood, but also by the swelling of the jugular veins, which manifests itself still more during the efforts of coughing.

When, finally, the carbonic-acid poisoning has reached a certain stage, the color of the skin changes from dusky to livid.

The cerebral functions undergo disturbance: the patients present pupillary contraction, have convulsive paroxysms and finally succumb after in some cases manifesting the peculiar kind of cerebral respiration known as Cheyne Stokes respiration.

Suffocative Catarrh in Children of the First Dentition.—All that we have just said applies especially to children above the age of two

years; in the very young child, in the nursling, the dyspnœa often takes on alarming proportions, under the form of sudden attacks of suffocation; children, however, that are feeble, the rickety and those suffering from wasting diseases, seem scarcely affected, cough feebly, evince a state of apathy, with small pulse, cyanotic aspect, and dyspnœa of a tranquil character, none the less dangerous for being calm. The dyspnœa gradually increasing passes unnoticed, till all at once the asphyxiating oppression manifests itself; the danger is imminent when the respiration becomes irregular and intermittent.

Suffocative Catarrh in the Adult and Old Person.—Suffocative catarrh in the adult may be primary. I have seen two instances in robust individuals, who after a seemingly trifling cold were attacked by suffocative catarrh attended with scarcely any râles, and died without presenting a trace of lobular pneumonia. It is the same with old men; if they be affected with any cardiac or vascular lesion, suffocative catarrh is not uncommon and is about sure to bring sudden termination to their life.

Auscultation. Fine or Subcrepitant Rales.—The congeries of symptoms which we have just analyzed so minutely under the head of dyspnœa is a faithful expression of the sufferings of the organism. Under such interruption of hæmatosis as characterizes capillary bronchitis, and especially that form which Laennec was the first to describe as suffocative catarrh, if at the same time that these functional disturbances take place, you perceive on auscultating one or both sides of the chest over a limited portion, or over a lobe, fine, subcrepitant non-metallic râles, whether or not there may be other râles accompanying, you may be almost certain of the existence of this disease. When once the minute bronchi are invaded, whether primarily or after the invasion of the large bronchi, as soon as these tubes are filled with a mucous exudation the conditions for the production of fine râles appear, for these indicate that during inspiration the walls of the bronchi detach themselves from the exudation. It is worthy of note that these fine râles of capillary bronchitis want completely thas metallic and resonant timbre which is heard only in the case of indurated or cavernous lungs. Hence the respiratory *murmur* remains always *vesicular* and cannot transform itself into bronchial respiration, except under the same conditions as give rise to resonance of the râles. The murmur may moreover entirely disappear in certain isolated regions, as happens when the afferent bronchi become impermeable to the air; it may, in short, appear and disappear by turns according to the degree of obstruction of the small bronchi.

Other râles, dry or mucous, of variable volume, may be heard in different parts of the chest, and mask the subcrepitant râles which belong to the capillary affection; but it should be borne in mind that while the large râles or the sibilant râles are disseminated without order, without regularity, the fine subcrepitant râles, on the contrary, have their seat of predilection in the infero-posterior regions of the lungs. This happens especially in little children, so that if those parts of the thorax are free from all morbid change, you may safely say that all the other segments are free as well. This predominance of fine râles in the posterior inferior part of the lungs imposes naturally on the physician the obligation to examine nursling children in a standing posture, and not recumbent on the abdomen—a position generally adopted in these examinations.

Cough and Expectoration.—Cough almost always exists in capillary bronchitis, but it must be remembered that it is far more promi-

nent a symptom when the inflammation extends to the large and medium-sized bronchi. We know, in fact, that the zone of excito-motor sensibility from which the cough irritation habitually emanates—the cough zone—is the mucous membrane of the large bronchi; the exudation moreover is more copious in these tubes, which constitutes another cause of irritation. The cough is generally painful, dry, and only rarely and tardily acquires the grave tone which indicates maturation of the exudation.

The expectoration is, in fact, very small in amount, and is at the same time viscous, transparent and poor in cells, as at the beginning of all forms of bronchitis. It scarcely ever acquires the character of muco-purulent sputa. When the sputa come from the small bronchi, they take on, according to Niemeyer, special properties. If you collect them in water, the sputa of the large bronchi float on the top of the liquid in the form of frothy muco-purulent globular masses. The secretion of the small-sized bronchi is destitute of air and has a tendency to fall to the bottom of the spit-cup, unless it remains attached to the frothy masses from the larger tubes; when once detached from the rest it presents the appearance of fine opaque filaments, which resemble somewhat the form of the small bronchi, and hang in the lower strata of the liquid.

Fever.—In many cases of capillary bronchitis the disease develops in an insidious manner and without the least perturbation. The children attract attention only by the difficulty and shortness of their breathing and by their cough. Fever, however, is never altogether wanting in the course of the disease, except perhaps in the case of adults; sometimes indeed the bronchitis breaks out all at once with a febrile movement; repeated chills open the scene, and in infants are accompanied with certain nervous (convulsive) movements, the temperature rises without presenting a regular curve, and it is not demonstrated that if it mounts above 39° C. (102° F.) it with certainty indicates pulmonary inflammation; one would not infer pneumonia from the invading march of the affection unless the thermometer should remain at 40° C. (104° F.) for several days.

In these conditions the pulse is accelerated and small; the fever always provokes digestive troubles, such as want of appetite, with furred tongue, sometimes vomiting; and alternatives of constipation and diarrhœa.

March of the Disease.—Whatever may be the train of symptoms, whatever the origin of the capillary bronchitis, it undergoes rapid evolution; three or four days suffice for it to return to the state of simple bronchitis, and in this event the danger is past; otherwise it progresses towards the lungs, where it produces complete obliteration of the bronchioles, or the phenomena of alveolar bronchitis, *i. e.*, of broncho-pneumonia.

Obliterating Bronchitis.

When the inflammation gains the lobular bronchi, their obliteration is certain, and there result: 1. besides the asphyxia, which is dangerous in proportion to the number of bronchioles invaded—2. Atelectatic collapse of the pulmonary lobules corresponding to the bronchioles invaded; 3. Emphysema of the lobules which remain sound; this indicates the excessive strain on the pulmonary tissue in these same lobules.

These lesions manifest themselves by an entire change in the auscultatory signs and by a complete modification in the percussion tone.

Auscultation. Blowing Breathing, etc.—As soon as the vesicular murmur is replaced by blowing respiration, and the râles take on

the resonant character, and when bronchophony is audible, you are to fear the occurrence of collapse and induration of the lung in an extent more or less considerable according to the number and coherence of the lobules which have undergone atelectasis under the influence of the obstruction of their respective bronchioles.

Percussion.—Percussion now gives new signs. While simple capillary bronchitis is unattended with the least modification of the percussion sound, the limit of the pulmonary tone tends to be lower in the thorax and to advance toward the median line, as soon as the lung in its middle and inferior lobes is exposed to the action of an excessive distending force, causing emphysema of over-strained portions; if this emphysematous condition extends, it may involve the lung covering the anterior surface of the heart, and even, on the right side, the supero-anterior surface of the liver.

Besides these signs of the emphysematous expansion of the lobules, revealing itself by exaggerated tympanism, you will shortly observe all the signs of an obscure *sub-tympanism*, which results from subsidence of certain lobules and often resembles the dull sound of hepatization; this is an indication of atelectasis which is still quite susceptible of complete and rapid cure. If the dullness persists, you have reason to fear that your patient is entering upon the phase of alveolar bronchitis or broncho-pneumonia.

Alveolar Bronchitis.

This is the classical broncho-pneumonia, which by its origin is linked to simple or infectious bronchitis, and which by the manner of its invasion presents the greatest affinities to true pneumonia: it is in fact a lobular pneumonia, whether disseminated or agglomerated. In the first case it is not distinguishable, from a semeiological point of view, from bronchiolitis, unless by the persistence, the length, and the gravity of the disease. In the second case (*i. e.* the agglomerated form), it is liable to be confounded with lobar pneumonia, from which it differs only in the absence of that cyclical evolution, so habitual in true or infectious pneumonia.

PART II.

CHAPTER VIII.

ACUTE PNEUMONIA.

GENERAL HISTORY—SUMMARY—PNEUMONIA.—DEFINITION.[1]

THE word pneumonia is easy to define: pneumonia is the inflammation of the pulmonary parenchyma; but the difficulty begins when you try to penetrate the sense of this definition and limit its application. The ancients comprehended under this denomination all the acute febrile affections of the respiratory apparatus. Auscultation taught how to separate inflammation of the pleura from phlegmasia of the pulmonary tissue itself, and the distinction has remained sufficiently clear in the majority of instances, despite a certain number of complex cases which seem still to justify to some extent the indiscrimination of the ancients. Later the physicians of the Hôpital des Enfants, Rilliet and Barthez, Legendre and Bailly, effected a new separation between frank inflammation of the lungs and the divers anatomical lesions described under the name of *fœtal state*, lobular congestion, splenization, and carnization. These latter morbid states were united under the head of broncho-penumonia. This mode of classification, sufficiently justifiable in the case of the infant from an anatomical and clinical point of view, has no longer the same importance with reference to the adult, and becomes a subject of dispute when you come to the pathogenic and etiological interpretation of these so-called broncho-pneumonias. Grisolle, in his remarkable treatise on pneumonia, introduced a new conception into the history of pulmonary inflammations by his separation of the secondary pneumonias from the primary. It was the etiological notion brought into the classification of the pneumonias, in opposition to the purely anatomical element. But this notion, founded on general considerations alone, and lacking a precise criterion, could result in only a rude outline of a classification. The truth of Grisolle's conception is nevertheless confirmed by the recent facts concerning the parasitic nature of diseases. It is from these data that we are henceforth to demand the necessary criterion which was wanting to our predecessors. It will not do, however, to be deceived respecting the real bearing of the results acquired the last few years. One may by the help of these new acquisitions sketch out a classification, he has not yet the requisite facts to enable him to fill in the outlines. What remains incontestible is that if you desire to establish new species of pneumonia, you must justify their existence by signalizing the presence of a special parasite, the real cause of the pulmonary lesion. For the acute pneumonias, as for the other affections of similar nature, the parasitic etiology must be the basis of

[1] Part II. was written with the collaboration of my Clinical Chief, Talamon.

every approach at etiological classification. It is not at all doubtful that to this special point the stress of scientific endeavor will in the future be directed. The ancient terms,—the denominations usually employed in clinical medicine and pathological anatomy,—must henceforth be regarded only as provisional guide marks, which every step in advance tends to render unnecessary, as a temporary scaffolding, to be removed as the edifice is completed.

Such are, indicated in broad features, the various data of the problem which the study of acute pneumonia actually brings before us. We will now endeavor to examine the subject in detail.

Pneumonia and Broncho-Pneumonia.

The writers of the XVIIth and XVIIIth centuries were obliged as conscientious observers to recognize the existence of at least two varieties of pneumonia; the one they called *peri-pneumonia vera*, simple frank pneumonia; the other they distinguished under the name *peri-pneumonia notha*, bastard pneumonia, false peri-pneumonia. This division, essentially founded on clinical observation, finds its justification in the anatomical researches of the first half of this century, and thenceforth it was chiefly pathological anatomy which emphasized the differences between primary pneumonia and that form which authors called broncho-pneumonia. We have already said that under this denomination authorities had included a certain number of lesions, the different appearances of which did not seem to agree with the notion of a process always identical in its evolution. It suffices to cast a glance over the works of physicians who have built up the history of broncho-pneumonia, to get an idea of the diversity of views which have been put forth on this subject.

Leger, who the first, in 1823, made a distinct separation of the pneumonia of infants from the primary pneumonias of adults, was struck by two facts: the symmetry or bilateralness of the lesions on the one part, and on the other the particular aspect of the pulmonary tissue in these cases, an appearance which he compares to that of the parenchyma of the spleen; whence the name which he gave to it, splenization. The writers who followed, Berton, Burnet, De la Berge, noticed the lobular disposition of the lesion, and its relations to bronchitis. In 1838 Rilliet and Barthez indicated a new distinct character, and described two varieties of pneumonia in the infant, the one primarily lobar, the other consecutive, lobular. But they were obliged by stubborn facts to recognize the existence of secondary lobar pneumonias; these were nevertheless in their view almost always broncho-pneumonias. Barrier admits three varieties; a disseminated lobular pneumonia, a generalized lobular pneumonia, and a pseudo-lobar pneumonia. This division corresponds to the entire congeries of facts, and from a descriptive point of view a better can hardly be found. But dispute was destined to arise on one capital point; the real nature of these pulmonary alterations.

Down to this epoch the different physicians whom we have mentioned had never entertained a doubt as to the inflammatory character of the lesions which they were describing. With Fauvel, and especially with Legendre and Bailly, the situation changes; capillary bronchitis dominates the scene; the pretended pneumonic lesions are only the mechanical consequences of the occlusion of the bronchi. The description so complete,

given by Fauvel, of the lesions of capillary bronchitis, had prepared the professional mind for this new conception. Legendre and Bailly, by showing the important rôle played by collapse of the lobules in infantile pneumonia, accomplished the revolution. According to them, the fœtal state and the disseminated or generalized lobular congestion, include the different anatomical appearances described under the name of splenization or lobular pneumonia. These are not inflammatory states. They found the proof of their view in the results furnished by insufflation of the parts affected. The atelectatic or congested lobules in fact recovered their normal appearance by this process, and only those lobules which resisted insufflation were regarded as inflamed and hepatized. But such resistance is rarely met with; true lobular hepatization is, then, a rarity. When it exists, it is only a variety of frank pneumonia, distinguishing itself from lobar pneumonia only by its lobular circumscription. The pneumonia has come to graft itself on the congestive lesions which accompany bronchitis; if there exist differences between the pneumonias of infancy and those of adult life, it is a matter of seat and extent, not of nature Such is the view of Legendre and Bailly.

This conception is very lucid and very seductive, and although the histological researches of Charcot and Balzer have led to another pathogenic interpretation of the broncho-pneumonic lesions, it is not demonstrated that this view is not, in a certain number of cases, the expression of the truth. However this may be, authorities were thenceforth divided into two parties; the one maintaining the existence of lobular pneumonia as a regular pulmonary inflammation, the other admitting only capillary bronchitis with mechanical lesions of the alveoli. Traube, Lebert and Vulpian range themselves in the first category. "Lobular pneumonias and splenization," says the latter in his thesis on the secondary pneumonias, "are phlegmasic hyperæmias which may undergo, within certain limits, the influence of gravity (relatively to their development and their situation) and thus participate in some of the characters of hypostasis." Hardy and Behier, on the other hand, declare themselves firm partisans of the non-inflammatory nature of the alveolar lesions. The inflammation does not overpass the finer bronchi; this inflammation brings on subsequently a congestion of the perilobular vessels, but the alveoli are not the seat of any phlegmasic process.

Limited to the disclosures of the macroscopic examination alone, the discussion might have been interminable. Taking as the type of pulmonary inflammation frank fibrinous pneumonia, such as Laennec described it, pathologists alleged as arguments against the inflammatory character of lobular pneumonia and the so-called splenization, the widely different appearances which these latter lesions present from those characterizing ordinary pneumonia, the smooth and even aspect of sections of the parenchyma, the absence of granulations, the extreme friability of the tissue, etc. Behier in particular strengthened this position by the examples furnished by the post-mortems of adults and old people, in whom the lobular hepatization is much more infrequent and much less defined than in the infant.

It was then that the aid of the microscope was invoked. The thesis of Damaschino (1867) marks the first stage in this new means of investigation. The histological lesion of lobular pneumonia is the proliferation of alveolar epithelium with production of pus globules; nevertheless in some cases Damaschino observed a veritable fibrinous exudation. According to him, then, broncho-pneumonia is without doubt an inflammation,

but an inflammation presenting the characters of catarrhal phlegmasia, dominated and disguised in part by the bronchitis and pulmonary congestion.

Catarrhal pneumonia—such seemed to be for a certain time the true name of broncho-pneumonia; this catarrhal pneumonia was quite clearly demarcated from fibrinous pneumonia; the one was characterized by desquamation and proliferation of the alveolar cells, the other by fibrino-plastic exudation. It was too willingly lost sight of that these catarrhal lesions are observed with the same general aspects in the first stage of fibrinous pneumonia, and that in fact between the splenization of lobular pneumonia thus characterized, and the engorgement of croupous pneumonia, the microscope shows no difference. It was forgotten, moreover, that Damaschino has signalized in several cases an exudation of fibrin in the nodules of catarrhal pneumonia, and that Virchow and Rindfleisch had observed the same. In fine, Charcot and Balzer have demonstrated the constancy of fibrinous exudation in what they call the peribronchial nodule.

The elementary lesion, then, was not sufficient to differentiate the two varieties of pneumonia. Fibrinous exudation and epithelial proliferation existed in both cases. Charcot sought and found in the disposition, in the topography of the microscopic alterations, the veritable characteristics of lobular pneumonia. It was in aaccordance with, and imbued with his ideas, that Balzer undertook the anatomo-pathological study of the infantile pneumonias; a study which led to the same conclusions as Charcot had formulated. In the view of these two pathologists, whose labors are summed up in the thesis of Joffroy, bronchitis of the small bronchi is the primordial essential fact in the development of the pneumonia; it is from the inflamed bronchiole that the phlegmasic process radiates. Lobular pneumonia is essentially characterized by the *peri-bronchial* nodule, which, on section of a lobule, is found to be constituted: 1. in the centre, by the bronchiole filled with a purulent exudation, and by the accompanying pulmonary arteriole, both the bronchiole and the arteriole being imbedded in a zone of inflamed connective tissue; 2. at the circumference, by one or several rows of alveoli containing fibrine filaments, intercrossing, with epithelial cells and leucocytes in their meshes. We have here the constant element which will be found every time that the inflammation shall have spread from the bronchus to the lobule, whatever may be the anatomical form of the lesion,—disseminated lobular, pseudo-lobar, or generalized lobular phlegmasia. At the periphery of the nodule you will see the congested alveoli filled with epithelial cells, more or less granular, and with pus globules; these are the splenized portions, in the midst of which the hepatized portions are scattered, like islands in the sea. (Charcot.) It is the lobular bronchus which directs the inflammation; which is, in a certain sense, the axis of the pneumonic process. The word broncho-pneumonia ought, then, as a consequence to be adopted as the preferable term to designate this variety of inflammation of the lungs.

In pursuing to its inmost seat, and in demonstrating the histological processes of the lobular pneumonias, the microscope may fairly be said to have constituted a group of pulmonary inflammations which have their starting point in the bronchial tubes. This work, perseveringly continued now for a score or more of years—has it given us any light on the mode of evolution, and on the processes of that variety of pneumonia called *frank peri-pneumonia?* All that we are able to conclude, is that

in this last group the bronchial element has only an accessory rôle, and that the distribution of the respiratory twigs cannot here be appealed to to explain the invasion of the alveolar parenchyma—what the simple naked eye examination, moreover, enabled us to affirm without any other proof. Must we invoke the intervention of the circulatory system to explain the topography of the lesions of frank pneumonia, after the example of Boerhaave, who admitted *à priori* two kinds of peri-pneumonia, the one developed in the territory of the bronchial artery, the other in that of the pulmonary artery?

There exist inflammations of the lungs of vascular origin, those, for instance, which are produced around emboli or thrombi of the pulmonary artery, but neither in their march, nor in their aspect, nor in their distribution, do these inflammations resemble frank pneumonia. As for the rôle of the nervous system from this special point of view, it cannot be invoked except in the way of hypothesis. One cannot in this regard draw any favorable conclusion from the experiments of which we shall speak farther on when treating of the subject of pneumonias by nervous action. What appears pretty certain is that the inflammation of frank pneumonia propagates itself from neighborhood to neighborhood, from alveolus to alveolus, from lobule to lobule, and even from one lobe to another, without having the least relation to the ramifications of the bronchial tree or to any vascular lesions. We must not be astonished if we find ourselves in reality less advanced as concerns the standpoint of the intimate evolution of frank pneumonia, than we are in our knowledge of the processes of broncho-pneumonia. During the time that the description of splenization and of lobular pneumonia was exciting so much dispute, the pathological anatomy of true peri-pneumonia remained as Laennec had described it. It was, for all the world, the type of frank inflammation; no one attempted to penetrate farther. The anatomical problem which excited such passionate interest, while it was a question of broncho-pneumonia, seemed resolved and unworthy of discussion when it had to do with frank pneumonia. Here it was the general symptoms of the disease, the march —so remarkable—with its abrupt commencement, with its sudden defervescence, which attracted the attention. The controversy pertained especially to the theoretical and analogical side of the affection, but its results were mainly the same as those attending the anatomical discussion of the lobular pneumonias; they ended in emphasizing still more clearly the difference separating frank pneumonia from the broncho-pneumonias.

The Doctrine of the Pneumonic Lesion.

According to Laennec, Andral, Grisolle, according to the majority of physicians who have written on this subject, frank pneumonia is a local affection, a primary inflammation of the lungs, and the general symptoms are in direct proportion to the extent, profundity, and the gravity of the pulmonary lesions. This idea was the natural corollary of the pathoanatomical disclosures and the stethoscopic studies of the commencement of this century. Our ancient predecessors, on the contrary, unable to follow by auscultation the progress of the lesion, seeing only its general effects, its march so frequently cyclical, made the pulmonary lesion which escaped their means of observation, subordinate to the general condition which alone was accessible to them, just as we do now for typhoid fever. They held that pneumonia was but the principal localization of a general disease, the *peri-pneumonic* fever.

After the discoveries of Laennec, the physicians of Montpellier were almost alone in maintaining the ancient doctrine. But little by little, since 1870, the pneumonic fever theory has been regaining partisans as well in France as in Germany. In 1873 Marrotte said: "There exists a disease identical by its causes, its general symptoms, its type, its duration, its critical terminations and even by its treatment, with simple synochal fever, from which it differs only by the presence of a local accessory affection, the pneumonia, which during the entire course of the disease plays the part of a simple phenomenon."

In Germany, Traube, Cohnheim, Jurgensen maintained the same view with certain modifications. Jurgensen thus sums up his opinion in the article on croupous pneumonia in Ziemssen's Cyclopædia: "Croupous pneumonia is a constitutional disease and is not dependent upon a local cause. The inflammation of the lungs is merely the chief symptom, and the morbid phenonena are not due to the local affection. We must admit a specific morbid agent. Croupous pneumonia, then, belongs to the group of infectious diseases."[1] At the last Medical Congress at Berlin (1884), the German pathologist repeated this proposition in almost the same terms. Here a confusion arises between two questions which are not by any means identical or inseparable, as the partisans of the pneumonic-fever theory seem to believe. Is pneumonia a general disease? Is pneumonia a disease produced by a specific agent? These are two subjects which are altogether distinct, and the demonstration of the second part of the problem by no means entails as a necessary consequence the demonstration of the first. It leads even much more logically, as we shall have occasion to show, to the weakening of the doctrine of pneumonic fever, and to the adoption under a somewhat different form, of the idea previously formulated by the French school, that pneumonia is a disease primarily local.

These two points ought then to be studied separately, and it is necessary to reject first of all the theory of pneumonic fever, as has been done by Hallopeau and Lepine; the one in an excellent critical review published in the *Revue des sciences médicales* for 1878; the other in his remarkable article entitled *Pneumonia* in the *Dictionnaire de médicine et de chirurgie pratique*, as we have ourselves done in our Lessons on the Infectious Pneumonias.

Let us, then, pass rapidly in review the arguments invoked in favor of their proposition by the partisans of the pneumonic-fever theory, and in particular by Traube, who, in the epoch when he wrote, had in view only the demonstration of the special point with which we are now concerned: "pneumonia is a general disease, and is not dependent on a local cause;"—the notion of the infectious or parasitic nature of the disease dating only from the last few years. Since the time of Traube, moreover, the arguments in defence of the view above stated have not changed, and it is always on the same phenomena—existence of prodromes, appearance of the fever before the local signs, persistence of the signs after the disappearance of the febrile element, sudden and critical defervescence of temperature—that authorities have relied in denying to pneumonia the character of a local affection.

1. Pneumonia, says Traube, has prodromes. That is true in many cases in an evident manner; the patients complain of general malaise, cephalalgia, nausea, sometimes even of epistaxis, several days before the

[1] Ziemssen's Cyclopædia, New York, 1875, vol. v., page 144.

pain, before the stitch in the side. Still more, we are convinced that the pretended instantaneousness of the commencement of pneumonia, surprising its subjects, as it is said to do, in a state of perfect health, is but a fact of tradition, and one knows the power in medicine as elsewhere of the traditional dogma. On the one hand, the patient, impressed particularly by the violence of the phenomena which accompany the chill, does not think it worth while to complain of any precursory malaise; on the other, the physician, imbued with the conventional notion, is content to accept the statement of the patient, which seems to him too natural a confirmation of the traditional view to need contradiction. But whether the prodromes be constant or not, whether they have a duration of several hours or several days, we cannot consent to find here a proof of the view that pneumonia is a general disease and is not dependent on a local lesion. There is no local phlegmasia whatever, not even a phlegmon of the hand, which does not necessitate a certain process of preparation, of germination, before giving rise to the violent disturbance of the nervous system which manifests itself by the chill. Between a prick, an abrasion of the finger, and the development of the phlegmon, there elapses a certain interval during which the person who has received the hurt experiences a general more or less pronounced malaise, vague pains, and headache before the outbreak of the usual chill. We have here, then, prodromes occurring apart from the local lesions, which are noticed or not, according to the degree of susceptibility of the patient's nervous system, and according as they attract or not the observant attention of the physician. Nobody has ever for this reason thought of making of subcutaneous phlegmon a general disease.

2. The fever may precede by from one to three days the pulmonary localization.—At what moment are clinicians agreed to recognize the inception of the pulmonary localization? On what signs do they rely in predicating one day something whose existence they would deny the evening before or two days before? Is it on the pain in the side? But the pain may be wanting all through the course of the disease, although the entire lung is nothing but a block of hepatization. Is it on the signs furnished by percussion and auscultation? But while the fever is perceptible from the very first instant, thanks to the thermometer, and the chill which ushers it in, the lesion is not accessible to our means of investigation till certain modifications of the pulmonary tissue are produced, such as congestion, engorgement, solidification. But on the one hand the genesis of these alterations cannot be instantaneous; on the other, however fine may be the ear, however delicate the finger of the observer, we know very well how little the stethoscopic signs or those furnished by mediate or immediate percussion are in relation with the real extent of the lesions. To produce the first physical signs which we perceive, it is necessary that the pneumonia shall have already invaded a notable extent of the pulmonary parenchyma. Is it too much to accord to this invading process one, two or three days, in proportion to the profundity of the original lesions, in order that it may attain the degree of extension necessary to be appreciated by our means of diagnosis?

3. The fever terminates by crisis the seventh, eighth, or ninth day.—This argument would tend rather to support the specific nature of pneumonia; hence it can be of no value in proving that the disease is primarily general. It will not do, moreover, to exaggerate the value of critical days, as a proof of the specificity of the lesion; else what will you make of pneu-

monias which terminate on the third, fifth, sixth day, or which are prolonged to the tenth, twelfth, and even fifteenth day? As the disease ordinarily ends in recovery, it must necessarily terminate on one day or another. "*Cuncta sublunaria,*" says Von Helmont,"*quae non sunt mutata, sunt saltem termino subjecta.*"

4. The local signs persist after the fall of the fever.—This is explained very naturally; since the lung is infiltrated with fibrine a certain time is necessary for resorption of the exudation. As for knowing whether the fall of the fever precedes the cessation of the inflammatory changes, or whether this cessation causes the defervescence, this is plainly impossible, as auscultation can give us only data of a gross kind concerning the physical state of the lung, and cannot in the least instruct us as to the intimate modifications which regulate the phlegmasic process. The argument, then, is nothing but an hypothesis, and in a certain number of cases the attentive study of the physical signs enables us to perceive the falseness of the hypothesis. Grisolle, who could not, it is true, measure the fever by the thermometer, says in his Treatise on Pneumonia: "In 192 patients, I have in ninety-four seen a considerable diminution in the febrile symptoms coincident with an amelioration in the condition of the lungs, as shown by the stethoscopic phenomena; the physical signs becoming more normal simultaneously with improvement of the general symptoms. In seventy-two other patients a notable diminution in the febrile movement has preceded by one or more days the favorable changes as revealed by auscultation and percussion. In twenty-six other patients there was a quite manifest return to the normal with respect to the phenomena of auscultation, at the same time that the fever continued in almost its original intensity."

Lepine moreover affirms that in many cases he has been able to announce the advent of resolution by the physical examination of the lung, several hours before the thermometer had indicated defervescence. In fine, according to Thomas, not only is it not rare to see resolution precede the crisis, but percussion enables one even to detect this resolution some time before defervescence, by the tympanitic character which the dullness then takes on.

5. Pneumonic fever has the greatest analogy to erysipelatous fever; it resembles it by its march, by its termination, by its recrudescence.—We do not dispute this analogy. But this is not a valid argument in favor of the essentiality of the fever of pneumonia, for it would be necessary at once to show that erysipelas is itself a *fever*, and that the febrile phenomena are not under the dependence of the local inflammation—a demonstration which it seems to us cannot at the present day be made.

The arguments of Traube in favor of the existence of pneumonic fever independent of local pulmonary lesions, do not, then, stand criticism. No one of them leads to the admission that pneumonia is a general disease and is not dependent on a local cause. Let us examine now Jurgensen's arguments, which leads us to the second question:—Is pneumonia an infectious disease?

The geographical distribution of croupous pneumonia, says Jurgensen, is absolutely different from that of catarrh and of bronchitis; these two last diseases increasing in frequency in proportion as you advance from the equator to the poles, while the number of pneumomic cases remains about the same in all latitudes. The different seasons of the year, moreover, do not influence alike the frequency of pneumonia and that of bronchitis and pleurisy. Pneumonia cannot be engendered by the ordinary causes of inflam-

mation. It needs, just as does typhoid fever, a special determining cause. During the course of the disease there is no constant relation between the local symptoms and the febrile phenomena. No affection due to a local lesion presents a cyclical march so clearly defined as does croupous pneumonia.

The above argument needs to be divided into two parts. Only the last clause has reference to the essentiality of the pneumonic fever, and yet the argument founded on the cyclical march of the pneumonia, as we have seen before, ought to be the rather invoked in favor of the specific nature of the disease, than in favor of the essentiality of the fever; we shall not concern ourselves farther with this point. As for the other proposition, that the fever is not conformable to the lesion;—supposing it to be absolutely true in all cases, what does it prove in favor of the essential and primary character of the fever? Do we not know that this is true for a good many other affections? Do we not see a simple quinsy sore-throat, from cold, provoke in a child an intense fever, and, on the other hand, vast phlegmons in a diabetic or cachectic individual undergo their evolution without raising the temperature above several tenths of a degree? Would one say on this account that angina *à frigore* is an essential fever, and that the phlegmon cannot provoke fever except when it is the local expression of a general disease? That certain pneumonias of little extent and gravity are accompanied by a considerable elevation of the temperature, that others involving a large area are almost apyretic, is a clinical fact which is indisputable; but it seems to us that one can search and find the explanation elsewhere than in the hypothesis of an essential fever. Moreover, to go to the heart of the matter, is Jurgensen's statement the rule? Clinically we cannot appreciate the extent of the pulmonary lesion except by auscultation. But, we repeat, this method of determination is absolutely insufficient; to judge of the real extent of pneumonia by the limited focus of crepitant râles perceived in a part of the thorax, would be to commit a grave error. The fever may then appear out of proportion to the physical signs noted, when in reality the lesion which escapes our means of observation might suffice to explain it. On the other hand, one may in a great many cases be able to assure oneself that the exacerbations of the fever are in close dependence on an extension of the lesion. In fine, do we not sometimes see the invasion of the other lung keep up and prolong the febrile phenomena beyond the regular cyclical term of the fever? The fever, then, seems to us perfectly in relationship with the local state; if in some instances there seems to be want of correspondence, we should look for the reason in circumstances connected with the trophic state of the subject, the degree of nervous susceptibility, the age, the sex, etc., and not feel ourselves obliged to refer it to the essentiality of the malady. The other arguments of Jurgensen, relate to the demonstration, not of the doctrine of peri-pneumonic fever, but of the specific infectious nature of frank pneumonia. We freely accept from this point of view, the proofs drawn from the geographical and seasonal distribution of pneumonia. We shall have to return to this when treating of etiology. It is necessary nevertheless to bear in mind that arguments of this character, founded on statistical considerations, are always to be accepted with reserve and caution, and cannot of themselves bring conviction. It would probably be easy to oppose to the statistics of Jurgensen other statistics absolutely contradictory; the exact value attributed to the terms pneumonia, bronchitis, pleurisy, varying evidently according to countries,

according to epochs, and, for a stronger reason, according to the preconceived notions of statisticians. It is not the same as regards the proved inability of divers irritant agents, physical or chemical, to produce experimentally simple frank pneumonia in animals. This fundamental fact merits a more particular study, for it suffices of itself to establish the specificity of the disease.

CHAPTER IX.

EXPERIMENTAL PNEUMONIAS.

Experimental Pneumonias of Nervous and Mechanical Origin.

The experimental physiologists, in their experiments on section of the pneumogastrics, were the first to signalize the existence of pulmonary lesions consecutively to this operation.

But their discussions of these facts concerned rather the mode of production of these alterations than their nature. Some considered them as vascular troubles, due to vaso-motor paralysis; others referred them to the loss of the sensibility of the bronchial mucous membrane, and to accumulation of mucosities in the bronchi. Traube, who was the first to notice the resemblance of these lesions to those characterizing lobular pneumonia, proposed the theory which has obtained the most partisans; section of the pneumogastric, in his estimation, has but an indirect rôle, namely, in causing paralysis of the recurrent nerves, and consequently anæsthesia of the laryngo-bronchial mucosa: the introduction of liquids from the mouth and alimentary particles into the larynx no longer producing cough by which these foreign bodies are expelled, they remain in the bronchi and penetrate even to the alveoli, provoking inflammation. In fact by injecting into the respiratory passages water holding in suspension the débris of aliments, Traube succeeded in determining the same alterations in animals whose pneumogastric nerves had not been cut.

We have no occasion to enter into this purely physiological discussion of the rôle of the pneumogastric in the production of certain pulmonary lesions. It is known that Genzmer, in sectioning the vagus above the origin of the recurrent nerves, and in thereafter warding off the effects of paralysis of the larynx and the introduction of liquids from the mouth, did not the less obtain the same results, which cannot here be attributed to traumatic irritation of foreign bodies. According to Michelson, who has made a comparative study of the effects of section of the recurrents and of the vagi, the consequences are not absolutely the same. Section of the pneumogastrics determines lesions which are almost immediate and are seated for the most part in the middle and inferior portions of the lung. The lesions consecutive to section of the recurrents are more tardy, and are produced more especially in the bases. The section of the recurrents, which allows in the hare a longer survival, is now employed by experimenters in preference to section of the vagi, which causes too rapid death. This is the method which has been put in usage by Friedländer, Frey, Charcot, in their researches on experimental pneumonia. In whatever way they may be brought about, in no case does the aspect of these lesions resemble the alterations of frank fibrinous pneumonia. Genzmer has made a great number of sections of the pneumogastric, in order better to ascertain the modifications in the pulmonary tissue which this operation

determines. According to his observations, what is especially noticeable in such cases, is the sanguineous engorgement and the œdema; in the midst of the congested and œdematous parts, you find, especially in the upper lobes, certain indurated nodules constituted by gray hepatization. The bronchi are filled with an abundant muco-pus in which you recognize, by the aid of the microscope, leucocytes and alimentary particles; the mucous membrane is red, turgescent and inflamed. These lesions are the same as those Traube indicated, the same as were seen by Mendelsohn, Friedländer, Vulpian, Frey, Charcot and all physiologists who have repeated these experiments. We should add to these lesions, an emphysematous swelling of the anterior parts of the lungs and an atelectatic condition of other circumscribed nodular portions, the result of collapse, which, according to Arnsperger, constitute the principal alteration.

The microscopic study made by Friedländer, Frey, Charcot, and Gombault, confirms the difference already perceptible to the naked eye, between these lesions and those of true pneumonia. According to Charcot two facts are constant: the inflammation of the bronchial mucosa, with production of purulent mucosities, and the presence of foreign bodies of divers natures, such as vegetal cells, epithelial cells of the mouth, hairs of the animal, alimentary particles, not only in the bronchial tubes but even in the alveoli. During the first week you find parts of the lung splenized, in the midst of which are disseminated grayish and indurated nodules. The microscope shows in the splenized regions cells of pulmonary endothelium in the course of proliferation, and in the hepatized parts numerous leucocytes crowded together and encumbering the cavities of the alveoli; there exists besides, peri-bronchitis and peri-arteritis, with agglomeration of round cells encompassing these vessels and tubes. During the second week the hepatization gains the splenized parts;[1] the epithelial cells fill the alveoli, and the walls of these are infiltrated with embryonal cells. At the same time a great number of these elements of new formation are invaded by granulo-fatty degeneration, which explains the yellowish appearance of certain portions of the hepatized regions. In the dog, as in the hare, these alterations take on the same characters. Charcot has even noticed in the dog in the peri-bronchial nodules a fibrinous exudation like that which is seen in the broncho-pneumonias of the infant.

If we have given considerable importance to the pulmonary lesions following sections of the nerves, it is because they represent the type of pneumonias such as experimentation has been able thus far to produce, and because they disclose the intimate mechanism of the pulmonary inflammation. Constantly one finds in the bronchi and alveoli foreign bodies which came there from the exterior, or from the buccal cavity. As for the real lesion, these inflammations are lobular pneumonias, or broncho-pneumonias; as for the mechanism, they are, to use the German expression, pneumonias by *aspiration*. Whatever, then, may be the agent of irritation employed, from the moment that it is made to penetrate the bronchi, you have as a consequence, at first a bronchitis, then a broncho-pneumonia, never a frank pneumonia; this is in fact what all tentatives ever made in this direction demonstrate.

Bretonneau was one of the first to essay the provocation of pulmonary inflammations in animals by the disengagement of irritating vapors. He

[1] The term splenization is applied to the lung in the first or second stage of pneumonia, when its tissue resembles that of the spleen; it is a state of less condensation than hepatization.—Trans.

shut up some hares in casks in which were liberated the fumes of hydrochloric acid, and removed them just at the moment when they were about to perish.

At the necropsy he found both lungs strewn with spots of ecchymosis, and in the centre of the congested tissue nodules of lobular hepatization. These experiments have been repeated with ammonia and chlorine; nitrate of silver has been injected into the bronchi. Trasbot and Cornil employed turpentine. All these chemical agents determine the same results: bronchial irritation and nodules of broncho-pneumonia.

Other irritating matters have been put into use. Hohenheimer has injected purulent septic liquids, even putrefied blood, into the bronchi of dogs, on which he had practised tracheotomy. He has thus produced lobular pneumonia or pseudo-lobar broncho-pneumonia. These experiments may throw light on the pathogeny of pulmonary inflammations which supervene in tracheotomized infants, but not on that of true peripneumonia. It is the same with the tentatives of Wolfe, who injected into the bronchi of hares and guinea-pigs liquids containing the ordinary bacteria of putrefaction. Although this experimenter says that he obtained in one or two instances veritable lobar pneumonia, it is not at all doubtful that he had to do in those particular cases not with fibrinous pneumonia but with catarrhal pneumonia, or pseudo-lobar broncho-pneumonia.

Kuhn has approached the experimental question from another direction. Taking his departure from this theoretical notion that pneumonia is the dominant localization of a general infectious disease, he has essayed to reproduce it by introducing the agents of irritation, not into the bronchi but into the subcutaneous cellular tissue. He injected under the skin of adult hares pneumonic sputa, undiluted, or an aqueous solution of these sputa. Kuhn endeavored to show the infectiousness of those contagious pneumonias which he observed at the prison of Moringen, and of which we shall have occasion to speak farther on. Of eighteen hares thus inoculated, six got well after several days of febrile disease. Five succumbed in the two days which followed the inoculation; the lungs were congested and studded with ecchymoses. In fine the other seven hares died, or were killed on the sixth to the twelfth day, after having presented a very high elevation of temperature, diarrhœa and a paralysis, more or less marked, of the posterior extremities. The necropsy showed islets of lobular or lobar hepatization with serous adhesive pleurisy and sometimes a slight pericardial effusion. There was also observed some swelling of the spleen, tumefaction of the intestinal follicles and parenchymatous nephritis. A hare under the skin of which a bit of muscle was sewed, got well after a prolonged fever. Two others, in the subcutaneous tissues of which had been inserted a fragment of lung, and débris from pleural false membrane, succumbed; in these there existed no other pulmonary alteration but congestion with fibrinous pleurisy; the spleen was tumefied and one of the hares showed marks of parenchymatous nephritis. It is impossible to see in the results noted by Kuhn anything which approaches to the alterations of fibrinous pneumonia. The foci of hepatization and pulmonary engorgement, as well as the swelling of the spleen, of the kidneys, of the intestinal follicles, are lesions which are met with in all hares killed by septicæmia, whatever be the putrid matters injected under the skin.

Finally Heidenheim, having recourse to the agent commonly regarded as the most ordinary cause of pneumonia, has attempted in vain to induce

experimentally a fibrinous inflammation of the lung by the aid of cold. He has endeavored to imitate the alternations of heat and cold common to social conditions, by causing his animals to breathe air by turns cooled and then heated. He has never been able to determine a true fibrinous pneumonia. He has obtained tracheitis, bronchitis, and nodules of lobular pneumonia; in no case has he observed lobar inflammation or concomitant pleurisy.

We shall not speak here of traumatisms, the powerlessness of which to provoke frank pneumonia has been shown by experiments on animals performed by Jobert de Lamballe, as well as by the observations of wounds of the lungs in man. We shall have occasion to return to this in the chapter on etiology.

Hence neither chemical nor physical agents, nor putrid matters, whatever may be their source, nor the micro-organisms of putrefaction, nor even pneumonic secretions injected into the bronchi or inoculated under the skin, are capable of determining frank lobar inflammation, and the lesions of true peri-pneumonia. The constancy of these negative results leads much more surely than all the arguments drawn from the clinical march of the affection to this indisputable conclusion: *the intervention of a specific agent is a necessity.*

CHAPTER X.

EXPERIMENTAL MICROPHYTIC PNEUMONIAS.

Infectious Parasitic Nature of Frank Pneumonia.

The specific agent, Klebs believed himself to have discovered during an epidemic of pneumonia which he observed at Prague. He described it under the name of *monas pulmonale*, ranging it in the too hasty division of schiztomycetes in the class of *monadines*. This micro-organism had for its essential character, its mobility; isolated or coupled under the form of bi-monad, it presented a spherical form. From these monads are engendered small mobile rods of from two to ten millimetres in length, endowed with a slow oscillatory movement, susceptible of multiplying by segmentation. These rods, in their turn break up into motionless monads, each surrounded by a clear gelatinous zone, generally taking on the disposition of little chains composed of four or five immobile organisms.[1] Klebs declares that he has noted the presence of these monadines not only in the bronchial liquid and exudation of the hepatized parts, but also in the blood, in the liquids of the cerebral ventricles, in the kidneys and in the liver.

"In some cases," he says, "the monadines appear under the form of immobile granulations, but always we obtain by culture organisms possessing movement. The culture medium was white of egg; the monadines developed there rapidly and were then characterized by quite active movements."

The experiments of Klebs were directed by the same idea as those of Kuhn. Thinking that pneumonia is the result of the localization in the lung of monadines circulating in the organism, he introduced the morbid matter into the anterior chamber of the eye of his hares. As inoculative liquid he employed the bronchial secretion of pneumonic patients. In a certain number of cases the hares succumbed without grave lesions of the lungs. In four experiments, he observed simultaneously with multiple visceral lesions affecting the kidneys, liver, heart, a more or less extensive pulmonary hepatization. This is the description given by Klebs in one of the experiments which he regards as the most decisive: "The lungs were voluminous and very firm in places; on the left side almost the entire lower lobe presented this abnormal consistency. The indurated parts were reddish brown on the surface. On section they presented congested patches, and anæmic points mottled with granular and yellow colored zones. The arterioles of this indurated portion were full of clots visible to the naked eye; the rest of the pulmonary tissue was congested and œdematous. The pericardium contained a great quantity of pus; on the tricuspid valve were found little deposits of yellowish gray color, resembling the vegetations of vegetating endocarditis. The heart muscle was also streaked with

[1] Klebs, Beiträge zur Kenntniss der pathogenen Schizomiciten. Arch. f. Path. Exp., 1877, Bd. IV.

little yellowish points. The microscope showed rods and granulations in great numbers in the cardiac deposits, and in the clots of the pulmonary arterioles. These same organisms were found, but in less abundance, in the fibrino-cellular contents of the alveoli, in the hepatized regions."

Thus Klebs described as the parasite of pneumonia, certain roundish mobile micrococci elongating themselves into rods. None of these characters pertain to the veritable pneumonic coccus, and it is impossible to admit that Klebs has isolated or even seen the real parasite of pneumonia. As for his experiments, they differ from those of Kuhn only in the observation in different organs of micrococci and of bacteria, which the latter did not look for. The lesions produced by Klebs' monads are those of experimental septicæmia in general; for want of other proof the purulent effusion of the pericardium, and the yellow spots noted in the myocardium, demonstrate that this was only a case of pyæmic and septic infection.

Eberth described in 1881, in the false pleural membranes of a pneumonic patient that died from cerebral meningitis, certain round micrococci, isolated or in colonies.[1] Koch, in a case of secondary pneumonia, found also certain micrococci in the capillaries of the lungs and kidneys; he was the first to indicate their oval form.[2]

Friedländer,[3] in eight autopsies of frank pneumonia, noted the presence of micro-organisms in the laveolar exudation, in the fibrinous plugs of the bronchi, in the pulmonary lymphatics, which were (as it were) obstructed by them; these micrococci had an ellipsoid form; they were joined two by two, or arranged in the form of chaplets; they formed crowded circles which stuffed the capillaries, and gave them a varicose aspect. This parasite seems, he says, to be the cause of pneumonia, but he could not affirm it.

At the Medical Society of Berlin, Leyden and Gunther announced the same year, that they had found in the living subject, the micrococci described by Friedländer. By means of a puncture made with a hypodermic syringe in the hepatized parts, they had been able to obtain—each in a well marked case of pneumonia—a certain quantity of fibrinous exudation which contained these organisms. In two other tentatives of the same kind, Leyden was not so successful, not being able to find the parasites.[4] Gunther, moreover, called attention to the fact that the ellipsoid coccus stained by gentian violet was surrounded by an uncolored capsule.

Matruy sought for the parasite in pneumonic sputa. In sixteen cases he found micrococci of elongated form in considerable abundance, and always surrounded by a very transparent capsule. He adds, it is true, that he has met with these same organisms in other than pneumonic subjects. According to Spina, they may even be found in phthisical patients; but Matruy concludes that they exist especially, and in largest quantity, in pneumonic sputa.[5] Salvioli and Zastein make no mention of a capsule; in fourteen cases they noted in pneumonic sputa certain ovoid but mobile micrococci; they found these same parasites in the blood of their patients, and even in the serosity of blisters.

Griffini and Cambria, in their researches on pneumonic sputa, did not

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[1] Eberth, Zur Kenntniss der mycotischen Processus. Deutsch. Arch. f. Klin. Med., 1881.

[2] Koch, Mittheilungen aus dem Kaisir. Gesundheits. Amt., 1881.

[3] Freidländer, Die Schizomyceten bei der acuten fibrinosen Pneumonie. Virchow's Arch., 1882, Bd. 87.

[4] Soc. de Med. de Berlin, Séance du Nov. 20, 1882.

[5] Matruy, Ueber Pneumonie-Koppen. Wienner med. Press, June, 1883.

find the oval cocci; they described certain long bacteria from two to three thousanths of a millimetre in length, slightly constricted in the middle, and rounded at their extremities. These bacteria, inoculated in hares, did not produce pneumonia; hence these two pathologists did not regard these microphytes as etiological.

The different researches to which we have just referred have but a relative value in demonstrations of the parasitic nature of pneumonia. They establish only the presence of microphytes in the exudation or expectoration of pneumonic patients. But, micro-organisms of different varieties abound in all pathological products. Moreover the description of the pathological experimenters does not agree; some indicate a round coccus, others an oval coccus, others an elongated bacterium with swollen extremities. Some affirm the mobility of the parasite, others its immobility. To some of these observers, the principal characteristic was an uncolored capsule; others do not speak of this envelope, which in the case of still other observers, was met with in the sputa of patients that were in no sense pneumonic. Nevertheless the greater number seem to agree on the ellipsoid form of the micrococcus indicated by Koch and Friedländer. But the experimental demonstration by the culture of the microbe and by inoculation, remained still to be made.

This demonstration was undertaken simultaneously in Germany and in France by Friedländer and Talamon. Friedländer was the first to communicate to the Medical Society of Berlin, at the meeting of November 19, 1883, the result of his experiments. According to him, what essentially characterizes the pneumonic coccus, is the capsule which surrounds it. This capsule is to such an extent the essential element, that for the German microscopist it predominates over even the form of the microbe, and Friedländer gives up the ellipsoid form of the coccus as of no consequence. The characteristic of the microphyte is not its form but its envelope. In fact he says expressly: "The capsule always takes the form of the micro-organism, if this is round, the capsule is round; if it is elliptical the capsule is elliptical." In fact, in the plates appended to his memoir, published in the Fortschritt der Medicin, you will see represented, not only round and oval cocci, but even elongated rods surrounded with the capsule.[1] In the discussion which followed his communication, Friedländer repudiated all assimilation of his parasite to the coccus described by Salveoli and Zastein. "The particular characteristic of my micro-organisms," says he, "is the indisputable presence of a quite distinct capsule."

Nevertheless it is a fact which ought to have given occasion for reflection to Friedländer, that the microbes when cultivated have never presented this capsule according to him so characteristic. This Friedländer himself acknowledges. The culture made in gelatine peptone by the method of Koch gives round and often elliptical cocci, but never cocci provided with a capsule. According to Friedländer these cultures have a quite peculiar aspect. On the surface of the gelatine a little elevation is formed from which a whitish substance extends downwards into the nutrient medium. This is what the German author speaks of as a *nail-shaped culture* (*en forme de clou*). The coccus develops in the ordinary temperature of the room and does not liquefy the gelatine. Here again Friedländer is mistaken in giving this aspect of the culture as peculiar to the pneumonic microbe; the nail form, then, does not depend on the organism,

[1] Friedländer, Die Mikrokokken der Pneumonie. Fortschritt der Medicin, 1883.

but on the manner in which it is introduced into the gelatine peptone, namely, by means of a platinum wire.

However this may be, inoculations were made with the product of these cultures. Friedländer injected directly through the walls of the chest, by means of a Pravaz syringe, the microbes held in suspension in distilled water; the operation was performed on hares, guinea-pigs, mice and dogs. The hares, nine in number, remained absolutely refractory. All the mice succumbed in from eighteen to twenty eight hours after the injection. At the necropsy the lungs were found almost empty of air, and intensely congested, with nodules of red induration. Out of eleven guinea-pigs, six died with pneumonic lesions. Of five dogs, only one succumbed under these conditions. In another series of experiments mice were made to inhale a spray of a culture fluid containing the coccus in suspension. Many of them succumbed. "At the necropsy," says Friedländer, "we found developed typical pneumonia, and numerous encapsulated micrococci in the lungs, spleen, blood and fluid of the pleural cavity."

The communication of Talamon which followed his researches made in the laboratory of the Hôtel Dieu since 1882, was presented to the Anatomical Society November 30th, 1883, several days after the appearance of the memoir of Friedländer. Talamon does not describe a capsule as characteristic of his microbe; according to him, it is the form which specializes the pneumonic coccus. Seen in the fibrinous exudation, the microbe has an elliptical form like a grain of wheat. Cultivated in a liquid medium, such as an alkaline solution of extract of meat, it lengthens out, becomes thinner, and takes on the appearance of a grain of barley. It is on account of this special appearance that Talamon has proposed the name of *lanceolated coccus*. This organism is met with in the pneumonic exudation, whether this be examined after death or withdrawn directly from the living subject by means of a Pravaz syringe plunged into the hepatized parts. Once only, out of twenty-five cases, has he found it in the blood of a patient at the point of death.

Experiments of inoculation have been made on twenty hares, two guinea-pigs and four dogs. The injection of culture liquid, made through the skin into the pulmonary tissue of the two guinea-pigs, was without result. The dogs were absolutely refractory. As much as three syringefuls of the culture liquid, swarming with micrococci, was injected into their lungs, and this for three or four days in succession, without any effect. One dog only presented for twenty-four hours a rise of temperature to 41° C. (105½° F.) but the next day the animal had regained its usual health. In the case of the hares, the inoculations were made in two ways. Six hares were inoculated under the skin of the thigh. None of them presented either local phlegmons or pulmonary trouble of any sort whatever. Twenty hares were inoculated by direct injection into the lungs of culture liquid obtained from the coccus of the pneumonic exudation of human patients, or the blood of hares that had died of pneumonia. Four of the hares got well, after remaining sick for four, five and six days. The malady was attested by the elevation of temperature noted day by day, the crisis of recovery, by the fall of the fever. The temperature indicated by the thermometer before the injection, stood between 39.6° and 40° C. (102° and 104° F.), it rose after the injection and the days following to 40.8° and 41° C. (105° and 106° F.), to fall afterwards to 39.6° and even 39° C. (102° and 101.5° F.).

Of the hares which succumbed, two presented no alterations of the

lungs, which were scarcely congested, but there existed a fibrino-serous pleurisy and an extensive fibrinous pericarditis. In eight cases were noted the lesions of fibrinous pneumonia, such as is seen in man, occupying sometimes all of one lung, sometimes the middle portion or inferior lobe. The pneumonia coincided in every case with a pleurisy of the same nature and in most cases with fibrinous pericarditis. Nowhere was found any suppurative inflammation. The blood of the hares, as well as the fibrinous exudation, contained elliptical cocci in abundance. The culture of fragments of the diseased tissues in broth made from extract of meat, constantly reproduced the characteristic lanceolate microbe, but smaller than its original dimensions in the human subject.

The somewhat brief description of the lesions obtained by Friedländer in his inoculated mice, may leave some doubt in the mind; for the presence of nodules of induration in the congested lungs does not suffice to characterize fibrinous pneumonia. But the lungs of hares presented by Talamon to the Anatomical Society in substantiation of his communication, constitute specimens of true pneumonia which leave no room for doubt or dispute. "It is by no means an affair," he says, "of nodules of broncho-pneumonia or of congestion such as we habitually observe in hares dead from septicæmia, but of a veritable lobar fibrinous pneumonia with pleurisy or pericarditis of the same nature." "The naked eye examination as well as the microscopic examination, showed no difference between the lesions produced in the hare and the pneumonia of man. The inflamed lung was heavy, dense, weighing three or four times as much as the healthy lung; it presented a yellowish black color; it was friable under the finger, and sank when put into a vessel of water; in the bronchi little fibrinous moulds were observable. Under the microscope the alveoli appeared full of fibrine filaments intercrossing and matted together. The nuclei of the intra-alveolar spaces were in process of proliferation; the vessels were full of coagulated fibrine and blood globules."

Afanassiew has repeated the experiments of Friedländer and Talamon in the laboratory of Prof. Cornil. By culture in gelatine peptone of pneumonic exudation taken from a cadaver, he has obtained two kinds of organisms, certain roundish micrococci of large and of small dimensions, and a quantity of ovoid cocci which correspond to the microbes described by the two experimenters of whose labors we have given a summary. He does not attribute any importance to Friedländer's capsule, and here he is supported by Cornil. The round micrococcus inoculated or injected in animals has not produced any effect. With the product of culture of the ovoid coccus, an injection was made into the lungs of guinea-pigs which resulted in a double sero-fibrinous or purulent pleurisy; the lobe corresponding to the seat of puncture, and sometimes the neighboring lobe, were in a state of red hepatization. Rarely a sero-fibrinous pericarditis was noted. The blood taken from the ear during life contained almost always diplococcus.

In other cases the injection was made in the jugular vein, it gave rise sometimes to peritonitis, sometimes to pleurisy. The exudation contained a great quantity of ovoid microbes.

Experiments made with rats have given similar results; the rats succumbed more quickly than the guinea-pigs. In the case of dogs the injections did not determine any lesions sufficient to produce death. After the injection of a cubic centimetre and a half of the product of the culture

of the ovoid microbes, the dogs had some fever the first day, but they were well again on the second or third. After injection of two cubic centimetres, the symptoms of pneumonia were well marked. Afanassiew has noted a temperature of 41° C. (105.5° F.) and tubular breathing on auscultation; but at the end of forty hours the dog commenced to get better. It is necessary to kill the animal on the second day in order properly to estimate the alterations produced. You find then a marked hepatization, and the ovoid coccus in small numbers in the pneumonic exudation and pleural liquid.

It results from these different labors that the experimental demonstration of frank fibrinous pneumonia may be considered as accomplished. This pneumonia may be reproduced in animals, but this reproduction is impossible with ordinary irritant agents. In order that the characteristic lesions may develop, it is necessary that a specific agent, a special microphyte, shall be brought in contact with the pulmonary tissue, and there multiply. Ordinary frank fibrinous pneumonia, then, is an infectious disease, not in the sense of being a general disease, but in the sense of being a parasitic microbic disease. It is an inflammation primarily local, as Grisolle and Andral taught, but it is besides a specific inflammation produced and characterized by a definite microbe. It remains local as long as the parasite does not overpass the limits of the pulmonary parenchyma; it is then simple pneumonia. When it diffuses itself and becomes general, invading the neighboring organs or penetrating the general circulation, whether by way of the lymphatics or blood-vessels, it becomes infectant pneumonia.[1]

[1] Afanassiew and Cornil, *Soc. biol.*, 1884, and Cornil, Leçons professées à la Faculté de Medicine pendant les premiers semestres de l'année 1883-1884.

CHAPTER XI.

CAUSATION OF ACUTE PNEUMONIAS.

Primary Pneumonias and Secondary Pneumonias.

We have said that good etiological considerations require that henceforth the establishment of species of pneumonia shall be based on this necessary datum—the discovery of the parasite peculiar to the disease. Just at present this desideratum is not realized except with reference to one species, the kind of pneumonia called frank fibrinous pneumonia. From an etiological point of view we must recognize two great classes of pneumonias: primary and secondary pneumonias. But it is important to define the meaning of these terms. Grisolle, who was the first to establish this distinction, gives this definition: "Primary pneumonia is that form which smites a person suddenly, in the midst of perfect health. Pneumonia, on the other hand, is called *secondary, consecutive, symptomatic,* when it develops in the course of an antecedent morbid state more or less grave, which has acted the part of exciting cause, or at least of predisposing cause, and which moreover impresses on the intercurrent disease such modifications that the physiognomy of it is altered or completely transformed." Faithful to these premises he ranges in the category of secondary pneumonias, as well those which supervene in the course of diabetes, cancer, or cirrhosis of the liver, as those which complicate measles, small-pox or typhoid fever. Clinically, this division, so understood, can be maintained. Etiologically we cannot confound rubeolic or diphtheritic pneumonia with the pneumonia which develops in a gouty or diabetic person. For us, whether pneumonia appear in a healthy individual, or in an individual affected with an organic or chronic constitutional malady, it is still primary pneumonia, provided that it corresponds to the type of frank fibrinous pneumonia caused by the ellipsoid or lanceolate coccus. The secondary pneumonias are those which develop in the course of general infectious diseases capable by themselves of provoking inflammation of the lungs. These pneumonias correspond generally to the anatomo-pathological type of the broncho-pneumonias. Without doubt there exist complex cases, as for instance the pneumonias which appear in the course of typhoid fever or influenza; we shall have occasion later to discuss these facts. It is not, on the other hand, proved that parasites, other than the elliptical coccus, can determine a fibrinous inflammation of the lungs. But in a general manner, and in the actual state of things, the distinction which we have established seems to us (provisionally at least) to be a very near approach to the truth, and the name of primary pneumonia ought to be accorded to every form of pneumonia characterized by the presence of the lanceolate micrococcus.

The Parasite of Primary Frank Pneumonia.

We will now study the characters of this microphyte. We have seen that Friedländer, after having assigned to it an elliptical form, in his last memoir has abandoned this characteristic, to make of the capsule which envelops it the essential element; this capsule may be oval, round or very much elongated, according as the microbe itself is elliptical, round or rod shaped. This is to deny all value to the very form of the microphyte. He even describes empty capsules of the coccus; these capsules are formed, according to him, of mucine, or some similar substance. They are in fact soluble in water and dilute alkalies, insoluble in acids. In fine, they stain with aniline colors.

This is the process indicated by Friedländer for bringing out in full relief the envelope of the micrococcus: "With gentian and fuchsine violet you obtain in a very short time a well pronounced staining of the capsule, nevertheless the strong coloring of the fundamental substance renders often the image of the capsule indistinct. If you then treat the preparation while on the glass slide with alcohol, the capsule loses its color, the micrococcus deeply stained appears surrounded with a clear areola, which corresponds to the capsule. It is under this form that Gunther first saw and sketched it. It is necessary to employ the gentian violet in solution not in pure water, but in aniline water, in order that the stain of the capsules may resist the action of alcohol. The method which we recommend, then, consists in staining the dry preparation in a solution of gentian violet and aniline water. The glass slide is then placed for half a minute in a watch glass filled with alcohol; the fundamental substance dries rapidly, while the capsules of the micrococcus retain longer the gentian violet. The preparation may then be examined in water, or after mounting in Canada balsam." Friedländer attaches such an importance to the capsule of the micrococcus that he declares that it never is wanting in acute fibrinous pneumonia, and that in all the other forms of pneumonia studied by him, he has only found cocci without capsules. Nevertheless we must again insist here that this capsule is absolutely without value. Talamon does not speak of it; according to him it does not belong to the coccus; it is the result of the mode of preparation and staining of the exudation. It is seen around different microbes in all the bronchial mucosities treated by the process of Friedländer. This is also the opinion of Professor Cornil. "We believe," he says (and here he alludes to his labors in conjunction with M. Afanassiew), "that the capsular appearance is of artificial origin, and that it is produced by retraction of the protoplasmic liquid around the micro-organism." Moreover Frænkel, at the Medical Congress of Berlin in 1884, declared that the formation of the capsule is not a constant phenomenon; that this capsule is seen in other schizomycetes; in fine that it is not possible to consider the capsule as characteristic of the micrococcus of pneumonia. Friedländer himself at the same Congress in his reply to Frænkel, has given the finishing blow to this famous capsule, in his recognition that that which characterizes the micrococcus of pneumonia is not its capsule nor its culture in nail form, but the sum of phenomena which it determines.

The question of the pneumonic micrococcus may then be considered as settled as far as *capsules* are concerned. It is the *form* of the microphyte which ought to be held as characteristic, joined to its pathological effects as determined by inoculation. This form, according to Talamon, (and

here he is in agreement with the first observations of Leyden and of Friedländer himself,) is elliptical. The microbe has the form of a grain of wheat, 1 m. to 4½ m.[1] in length by ½ m. to 1 m. in width, in the largest diameter of the ellipse. It is isolated, generally in pairs, sometimes in chains of fours. In the hare it is much smaller than in man. It is under this form that Talamon has seen it in the fibrinous exudation taken from the living subject by the aid of a Pravaz syringe plunged into the lung through the costal parietes, or obtained several hours after death; once only did he find it in the blood of a patient during the death agony. The exudation ought to be examined without previous staining; the coccus stains by the aniline dyes, and in particular by the violet of methylamine, but it becomes deformed under the action of the coloring matter, and tends to take on a roundish form.

Cultures.—The microbe may be cultivated in an artificial medium. Friedländer has employed Koch's method, namely culture in a solid gelatine medium. Talamon makes use of Pasteur's process, *i.e.* the employment of a liquid medium made with solution of extract of meat rendered alkaline by potassa. According to the latter, a sowing made with the exudation collected after death, gives only impure cultures. In order to have the coccus in a state of purity, you must insist on the exudation being taken directly from the living subject; and yet one rarely succeeds in thus isolating the microphyte. Friedländer, by his process, has nevertheless succeeded in cultivating the micrococcus with exudation taken from the cadaver. Cornil and Afanassiew have likewise cultivated the post-mortem exudation in gelatine peptone, but they have only once succeeded in obtaining immediately a pure culture of ovoid coccus.

Friedländer, as we have seen, gives as the particular characteristic of the cultivated pneumonic coccus, its nail-like mode of growth. Afanassiew has obtained the same appearance, but this is an aspect due wholly to the mode of sowing. You will observe it as well in the case of other organisms sown in a medium of solidified gelatine peptone.

According to Talamon, in a liquid medium, the coccus acquires more considerable dimensions; it may attain 2½ m. to 3 m. in length, to 1½ m. in breadth; it is then that it takes on the lanceolate form and sometimes one of its extremities becomes tapering.

We shall not repeat what we have said respecting the results obtained by the inoculation of these microphytes in animals. The elliptical coccus determines a true fibrinous pneumonia, identical with that of man. But two important questions present themselves here: Is this ovoid micrococcus the only organism in the pneumonic exudation? Is it the only organism capable of producing a fibrinous pneumonia? In his communication to the Anatomical Society, Talamon declared that he has not observed in all his pneumonic patients the same organism, and he adds that it would be of importance to know if this fact has to do with differences of *form* or differences of *species;* he describes the ovoid coccus as the microbe which he has most often found. On the other hand, the researches of Afanassiew show that by the culture of the pneumonic exudation, one may obtain three varieties of microbes: 1. a large round micrococcus, 2½ m. to 2 m. in diameter; 2. A little round micrococcus, 0.5 m. to 0.9 m. in diameter; 3. Ovoid microbes of 1 m. in length. In fine, it is certain that the microscopic examination of the alveolar exudation, always reveals

[1] Thousandths of a millimetre.

a great number of round cocci mingled with elliptical cocci; indeed, often, especially after death, the first are much the more numerous. But it is well not to lose sight of the fact that every inflammatory pathological product always contains round micrococci; that even in tuberculosis the characteristic bacillus is often lost, as it were, in the midst of swarms of cocci. The nature and the rôle of these microphytic forms still remain to be determined. The experiments of Cornil and Afanassiew seem to indicate that in pneumonia the round cocci have only an accessory action, or at least an action which is in no sense specific, since in no case has the inoculation of the large round micrococcus isolated by culture, ever produced the effects obtained with the ovoid coccus. Nevertheless, in two cases of grave pneumonia with pericarditis and vegetating endocarditis, Talamon affirms that he has isolated a microphyte quite different from the wheat-shaped microbe. This new parasite was formed of chaplets of round cocci—flexuous chaplets—very long, composed of from ten to twenty grains. It is cultivated pure under this same form. But this organism in chaplets of grains, cultivated and multiplied under this form, has produced by injection in the lungs of hares, fibrinous pleuro-pneumonias identical with the pneumonias caused by the ellipsoid coccus.

CHAPTER XII.

EPIDEMIC PNEUMONIAS.

History of Epidemics of Pneumonia.

We are still too little acquainted with the causes of microbic pneumonia to be warranted in attempting to formulate from what we do know, the data of a rational and scientific etiology. We ought actually to limit ourselves to noting the conditions which seem to favor the development of this disease in man. From this restricted point of view, the study of epidemics of pneumonia, at the same time that it furnishes a new argument to the support of the infectious and parasitic nature of the disease, discloses to us an assemblage of interesting facts.

Despite the persistency of many modern pathologists in regarding fibrinous pneumonia as a simple inflammation of the lungs, epidemics of peri-pneumonia have long been mentioned in the writings of our ancient predecessors, under the name of pestilential, malign, putrid, nervous, typhoid pneumonias. The first epidemic described by Shekius, which in 1348 devastated all Europe, is regarded by certain writers as one of these epidemics of pestilential pneumonia. From 1585 to 1621 a similar epidemic raged in the south of Europe. According to Jean Calle of Urbino, the disease was characterized by its contagiousness, by the rapidity of its fatal issue, which generally happened from the fourth to the seventh day, by the suppurative destruction of the lungs, and pericardial effusions, as noted at the autopsy. In 1688 the epidemic of Brisgau, described by Voerster, raged chiefly among the soldiers. The disease was contagious. As in the cases observed by Jean Calle, the lungs were found after death hepatized and infiltrated with pus, the sac of the pleura and pericardium full of hæmorrhagic exudation. The same characteristics distinguished the epidemic observed by Lancisi at Rome in 1708, by Sauvages a little later in the south of France, also the epidemic which broke out in Normandy in 1756, and which from 1756 to 1758 swept over France from north to south; the epidemic of Berne described by Haller in 1762, that of Rouen mentioned by Lepecq de la Clôture in 1768, and finally that of Pithiviers which prevailed subsequently.

But it is very difficult to get an accurate idea of the real nature of these epidemics. Suppuration and gangrene, not only of the lungs but of most of the viscera, are indicated as a constant phenomenon in the post-mortem records which have been handed down. But it is necessary to bear in mind that these two morbid processes do not belong to pneumonia, even of the epidemic kind, such as we observe at the present day. According to Laveran, the epidemics described since the XVIIth century and down to the middle of the XIXth century, may be ranged in the three following classes:—1. Epidemics connected with malarial fevers, noted by Lancisi and Gagliani at Rome, and Jean Calle of Urbino; 2. epidemics prevailing at the same time as eruptive fevers, (epidemic of Brisgau in 1688); 3. epi-

demics developing at the same time as scurvy. If you add influenza, with which was associated the epidemic of pneumonia observed in Paris in 1837, you will have the four principal causes which preside over and explain the development of the ancient epidemics. These were, then, for the most part *secondary* pneumonias, and they ought not to be confounded with the primary pneumonias which we have really in view. It is worthy of note, at the same time, that the descriptions of the medical historians are exact; pulmonary symptoms predominated in these epidemics, and it was really by the lungs that the patients died with such extraordinary rapidity in four, five and six days. Without doubt the ancients were wanting in the knowledge and means of auscultation, but all the accounts of autopsies agree in certifying to the hepatized, purulent, gangrenous condition of the lungs, while synchronously the pleura and pericardium were found filled with a turbid and sanguinolent serosity. We no longer witness at the present day those vast epidemics which ravaged entire provinces, and this is without doubt due to the hygienic conditions of our populations, which are assuredly better now than they were in the previous centuries, and there may be an additional reason pertaining to the different nature of the cause of the pneumonias of the present day. The observations which have been made of late years concern epidemics of much more limited extent; but they enable us, by this very fact, to obtain a more exact appreciation of the pathogenic conditions and symptoms of the disease.

It is in Germany, in America and in England that these localized epidemics have been observed and described. We should nevertheless not forget that in France, in 1838, Torchet gave a description of the epidemic of pneumonia observed by him at Noyers, which may serve for a type of all the village epidemics noted since then.[1]

We may class the local epidemics in four orders, as we have done in our Lessons on the Infectious Pneumonias:[2]—Epidemics of prisons and of barracks; epidemics of small towns; family or household epidemics; finally, epidemics of cities.

We must set aside as doubtful, for the same reason which made us set aside the peri-pneumonic epidemics of the XVIIIth century, the account given by Bryson in 1863, of the epidemic observed on board the English fleet in the Mediterranean, on which occasion the pneumonia was coincident with scurvy,[3] as well as the epidemic described by Hjaltelin in Ireland, which was an epidemic of influenzal pneumonia.[4]

Epidemics of Prisons and Barracks.—The most important prison epidemics are those of Christiania in Norway, of Frankfort in Kentucky, of Moringen in Hanover, as related by Dahl, Rodman, and Kuhn. At Christiania, out of a total of 360 prisoners (the mean number of those confined within the precincts of the jail), Dahl observed from the month of December, 1866, to the month of May, 1867, sixty-two cases of pneumonia; six of the prison wardens were also affected. During the whole course of the year 1866, but three cases of pneumonia were noticed among the convicts. A similar epidemic had been observed in 1847 in the same jail; 69 of the prisoners were smitten; but there prevailed at the same time an epidemic of scurvy.[5]

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[1] Torchet, Mémoires de l'Acad. de Méd. de Paris, 1838.
[2] G. Sée, Des pneumonies infectieuses. Un. Méd., 1882, nos. 76–78.
[3] Bryson, Soc. épidém. de Londres, déc., 1883.
[4] Hjaltelin, Epidemic pneumonia in Ireland. Edinb. Med. Journ., 1864.
[5] Dahl, Norsk. Mag. for Loegevid, Bd. XXII.

At Frankfort jail (Kentucky State Prison Hospital) there were also two epidemics; the one in 1875 made victims of 75 of the prisoners; the mortality was light and did not exceed eight per cent. The other epidemic the year following was much more severe; twenty-five patients out of ninety-eight succumbed. Rodman gives few details respecting the clinical march of the disease; he records simply that the physical signs and anatomical lesions were those of frank pneumonia. He notes the frequency of pleural lesions. The liver was engorged and full of black blood. He does not speak of the spleen. Pain in the side was often wanting, and the patients in many instances raised pure blood in large quantity. The intestines were not much affected, but the stools were horribly fœtid. The temperature rarely attained as high a point as in ordinary pneumonia.[1]

The narration of Kuhn is richer in clinical and anatomo-pathological details. At Moringen, as at Frankfort, two epidemics appeared successively; in that of 1875 eighty-five prisoners were attacked: in 1878 fifty-eight cases were recorded. The disease began with vague prodromes, general malaise, want of appetite, lumbar pains which lasted four or five days, then repeated chills came on, without pain in the side or expectoration. Rarely, however, the invasion was sudden with violent chill. It was not till the third or fourth day after the commencement of the fever that the ordinary local signs of pneumonia appeared. The march was then that of *migrating pneumonia*, that is, the hepatization was not fixed, and there were produced successively, several scattered foci of inflammation and consolidation. The disease was constantly accompanied with grave pleuritic symptoms, albuminuria and engorgement of the spleen. In two thirds of the cases there was diarrhœa. The thermometrical curve presented the typical aspect of the pneumonic curve; continuous fever with high maximum temperature of 104° to 106° F. and abrupt defervescence after the first seven days; but habitually there was a new ascension accompanying a new inflammatory outbreak, or else (as often happened) the febrile type became irregular, pleurisy or pericarditis taking the predominance over the pulmonary symptoms.

The first epidemic resulted in sixteen deaths, the second in eight. In the eight autopsies of 1878, an abundant pleuritic exudation was noted in seven, in five, a parenchymatous nephritis, in five, pericarditis, in four meningitis, in all ramollissement of the spleen. In five cases, the pneumonia was double; in three it was seated on the right side. The heart was almost always in a state of fatty degeneration. Twice only, Peyer's patches were slightly tumefied.[2] We might also cite the epidemic of Ausberg jail observed by Kerchensteiner.[3] In the first five months of the year 1880,

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[1] Rodman, Amer. Journ. of Med. Sc., 1876, vol. lxxi., p. 76, and New York Med. Record, March, 1875.

[According to Rodman's account in the Medical Record, (loc. cit., page 181), the temperature ranged from 103° F. to 105° F., pulse from 120 to 150, respirations from 30 to 50. Death was a frequent occurrence in less than twelve hours after the admission of the patients to the hospital. Rodman attributes a large part, in the causation of this epidemic, to the damp unwholesome cells in which the prisoners were confined—the prison was built on "made ground," in other words, on a reclaimed marsh; men were huddled together in cells that contained not more than 300 cubic feet each. The building was imperfectly ventilated. The occupation of the prisoners, most of whom were engaged in hackling, spinning and weaving hemp, may have had some predisposing influence.—Trans.]

[2] Kuhn, Deutsch. Arch. f. Klin. Med., 1878, and Berlin Klin. Woch., 1879, p. 552.

[3] Kerchensteiner, Bayr. arztl. int., Bl., 1881, XXIII.

one hundred and sixty-one prisoners were attacked with croupous pneumonia; forty-six died, while not a person among the employés of the house was smitten by the disease.

One might compare these jail epidemics with the epidemics of barracks or regiments, such as that of the garrison of Magdeburg, from October, 1873, to June, 1874, as reported by Grindler;[1] that of Wesel described by Kranz;[2] that which raged among the soldiers of the twenty-second regiment of New Brunswick, of which Welch gives a brief narration.[3] The most interesting of these accounts is that of Costello, Surgeon-Major Bengal Medical Department. The First Punjaub Infantry lost in a few weeks from thirty to forty men out of a strength of about five hundred and fifty; the Fifth Punjaub Infantry lost sixty men from a similar outbreak. Costello states that at the moment when the epidemic appeared among his troops they had just been marching through districts of Afghanistan known to be affected with cattle plague—contagious pleuro-pneumonia—from which source the soldiers seemed to have caught the infection.[4] The pneumonia invaded rapidly all of one lung, then gained the other; in every case there was poignant pain in one or both sides of the chest. The disease was accompanied from the first by complete prostration, with dryness of the tongue, dark sordes on the teeth, in a word with all the symptoms of the typhoid state.

Village Epidemics.—We have mentioned above, the epidemic observed by Torchet at Noyers in 1836. One hundred and twenty-four cases were observed by this physician in a village of several hundred inhabitants. The disease seemed to attack preferably infants and young people; out of 124 patients, 91 were less than twenty-five years old and 45 were under fifteen. The prodromes were more or less prolonged, but when the pneumonia was declared, the typhoid symptoms rapidly reached their maximum of intensity; profound stupor, continued somnolence or somnolence alternating with agitation and delirium; tremulous tongue, which was dry and shrivelled; bilious or black diarrhœa; burning fever—such were the symptoms attending the pulmonary inflammation. The latter was seated especially on the right side, and in two thirds of the cases it occupied the upper lobe. In the epidemic of Becherbach, reported by Butry,[5] out of 490 inhabitants, 20 were attacked by pneumonia in the space of a few weeks. In these 20 cases there were 9 deaths. The disease was limited to a small number of families, for the most part propagating itself from the circle of the relatives and neighbors of those first attacked. The typhoid symptoms were present in almost all the cases, and in five of these, pneumonia was accompanied with jaundice. The same characteristics distinguished the epidemic of Ober-Sitke, as described by Munnick and Holwede.[6] In this little village, which numbered scarcely four hundred inhabitants, fifteen cases broke out in a very short space of time, and in some instances, as many as three in the same family were attacked.

The epidemic observed by Penkert differs from the preceding by the

[1] Grindler, Deutsch. militar-arztl., Zeitsch, 1875, IV., 2.
[2] Kranz, Eulenberg's Vierteljahrsch. f. gerichtl. Medicin, XXXVII., 1.
[3] Welch, Med. Times and Gaz., December, 1869.
[4] Costello, London Lancet, Jan., 1881. You will find a description of a similar epidemic of pneumonia among the soldiers of the garrison of Valenciennes in the Journal de Médicine, December, 1857.
[5] Butry, Deutsch Arch. f. Klin. Med., 1881, XXIX.
[6] Munnick and Holwede, Berlin Klin. Woch., 1883, No. 23.

benignity of the disease. They are the characters of frank pneumonia which Penkert describes, and not those of typhoid or asthenic pneumonia. In a village of 700 inhabitants, 42 cases of pneumonia were observed in less than two months. Of these forty-two patients only two died; the invasion of the disease was always sudden, without prodromes; the congeries of symptoms was that of ordinary pneumonia. The duration of the disease never exceeded eight days. The twelve patients first affected were children who attended the same school. The others were adults, twenty-eight of whom, according to Penkert, acquired the disease by direct contagion; in four only indirect contagion could be suspected.[1] The epidemics described by Scheef, assistant of Jurgensen,[2] and by Seufft, belong to the same category.[3] The latter in the space of twenty-two days observed fifty-nine cases of pneumonia in a population of 1500 inhabitants. There were only five deaths.

House Epidemics.—"In our Lessons on Infectious Pneumonias" we reported three examples coming under our personal observation of this kind of partial epidemics. In the same family, husband and wife were suddenly taken at the same time with a violent chill, on getting up from the dinner-table. The next morning the signs of lobar pneumonia were recognized in both of them. The disease went through its course with typhoid symptoms of the most marked kind; complete prostration, profound adynamia, lips and tongue dry, tympanism of the abdomen, temperature very high, continuous sub-delirium. The wife died in this condition on the eighteenth day; the husband succumbed four days later. The pneumonia had commenced in the inferior lobe, and had rapidly invaded all the upper lobes of the lungs; in the wife the pneumonia was seated on the right.

In another family of five children, three were atttacked with pneumonia a few days apart; only one had symptoms which at first suggested typhoid fever, all three, however, got well. In fine, in a third family a man of sixty years of age succumbed after several days to an attack of adynamic pneumonia. His wife, aged forty years, healthy and robust, in her turn took the disease and died six days after her husband.[4]

Cases of this kind are to-day very numerous: they all resemble in their principal features the facts which we have just cited. You see the different members of a family, father, mother, children, smitten one after the other by the disease, which sometimes presents no more gravity than ordinary frank pneumonia, sometimes, on the contrary, kills in several days all those whom it attacks. In the memoir of Bonnemaison on the Malign Pneumonias, are recorded five cases of these little family epidemics; we quote the following:[5]

"An old man, seventy-six years of age, died on the sixth day of frank adynamic pneumonia. His daughter, who had come from a distant quarter of the city, and who had lavished on the patient the most assiduous attentions during the first three days of the sickness, was taken the day after her father's death with chills and malaise, and underwent a frank pneumonia, from which she recovered in about ten days. Her son, a volunteer in the Infantry, who had obtained a furlough of two days to visit his

[1] Penkert, Berlin Klin. Woch., 1881, Nos. 40 and 47.
[2] Scheef in Jurgensen, Beobach. aus der Tubinger Poliklinik, 1883.
[3] Seufft, Berlin Klin. Woch., 1833, No. 38.
[4] G. Sée, loc. cit. Un. médicale, 1882.
[5] Bonnemaison, Des pneumonies malignes Un. Méd., 1875.

parents, returned to his regiment, fell sick two days after his arrival in camp, and entered the military hospital. It appears from the hospital records that he was affected with pleuro-pneumonia, getting well in about a fortnight."

Lécorché and Talamon [1] relate a similar case. In a family composed of father, mother and a son sixteen years old, the mother died first, on the ninth day, of pneumonia. The son and the father, who during these nine days had scarcely slept from anxiety and watchfulness, eating little or nothing, both went out on an excessively cold day, to enregister the mother's death at the mayor's office. On returning home, they both had a violent chill at the same time, and were obliged immediately to take their beds. The father, who was cared for at home, succumbed several days after. The son, transported to the hospital DuBois, had double pneumonia of the most grave kind, with extreme typhoid prostration, delirium and albuminuria. The disease turned nevertheless on the thirteenth day, but the pneumonic inflammation was more than a month in undergoing resolution, and six weeks after recovery there was still a considerable quantity of albumen in the urine. The facts recorded by Wynter Blyth and by Hardwick are still more remarkable. In a certain family, a farmer attacked by pneumonia is nursed by his niece. She takes the disease in her turn and gives it to her husband.[2]

Hardwick also gives an account of a priest who was sick with pneumonia; one of his relatives came to see him and contracted the same disease, then a third person was taken with pneumonia after watching with the latter.[3]

In the epidemic described by Daly, six members of one family out of eight were attacked with pneumonia; the mother and grandmother succumbed, four of the children got well; the father and hired girl alone escaped.[4]

In the epidemic of which Patchett gives an account, an entire family composed of five members succumbed in less than a fortnight to pneumonia. The patients, it is true, were aged persons, but the observation is none the less curious. It was a case of four brothers and one sister, all unmarried, living in the same house. The eldest, aged 73 years, was the first smitten; she died in six days, on the 16th of January. The day after her death, her brother, aged 66 years, who the evening before had been taken with chills, complained of pain in his right side; he was found to be affected with pneumonia, which spread rapidly, and the patient succumbed on January 19th, three days after the invasion. On the 20th the two other brothers, aged respectively 63 and 64 years, were taken simultaneously with double pneumonia, which pursued its course with extraordinary rapidity. On the evening of the 22d both patients were dead. Finally the sister, aged 61 years, who had nursed her brothers during their entire sickness, and who seemed likely to escape, was attacked on the 23d with pneumonia on the right side and died on January 26th.[5]

In the account given by Ritter, seven persons living in the same house were attacked; three died. At the autopsy, in each case was noted fibrino-

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[1] Lécorché and Talamon, Études Médicales, Paris 1882.

[2] Wynter Blyth, Lancet, Sept., 1875.

[3] Hardwick, Pneumonia in Infectious and Contagious Diseases, Gaz. Méd. de Paris, 1876, No. 43.

[4] Daly, Contagious Pneumonia, Lancet, 1881.

[5] Patchett, Contagious Pneumonia, Lancet, Feb., 1882.

serous pneumonia with fibrino-purulent pleurisy; there was dilatation of the heart, hypertrophy of the spleen, swelling of the cortical substance of the kidneys, and absence of intestinal lesions. In all these patients the disease went through its course with grave ataxo-adynamic symptoms and continuous delirium. In one of the cases which ended in death, a roseolic eruption appeared on the body, coincident with labial herpes and punctiform ecchymotic spots on the lower extremities.[1]

The epidemic described by Muller pertains to a family of five persons who were attacked with pneumonia, one after another, in the space of a fortnight. The mother, aged fifty-three years, fell sick the first, on the 15th of November: the father, aged 64, was taken the 6th of December; the son, aged 18, the 7th of December; the daughter, who had come home to take care of her mother, was smitten in her turn on the 5th or 6th of December; finally the little boy, aged five years, was the last one taken, on the 10th of December. In all the march was that of pneumonia of typhoid type; except in the case of the child, the disease began by a chill and pain in the side; the expectoration was characteristic. There was profound asthenia with delirium, somnolence, dry tongue; the temperature nevertheless did not exceed 104° F. in the rectum. In both cases there was a complication of pleurisy; in the mother, endocarditis was noted. All the patients, however, recovered, and within the period of ordinary pneumonias, namely, with defervescence on the ninth day in one case, on the seventh and fifth in the others.[2]

We also desire to call attention to the observation of Bielenski; out of ten inmates of the same house, nine had in the space of a fortnight croupous pneumonia. Of these nine cases of pneumonia, eight were right-sided; all these patients recovered.[3]

Couldrey, Herr, Heitsch, Giornelli, and many other writers have published facts of the same kind.[4] All these observations are alike, and it would be useless to give in detail a larger number; those which we have just analyzed suffice to establish that pneumonia may behave like the most indisputably infectious diseases, diphtheria, or typhoid fever.

City Epidemics.—We shall not dwell on this class of epidemics. They are simply the repetition, on a large scale, of the little epidemics of prison or village. But the etiology is here less easy to elucidate than in the isolated epidemics. In small localities, in fact, pneumonia is disengaged from every other pathological influence. In the large cities, on the contrary, it is very rare not to see its prevalence coincident with that of other infectious and epidemic diseases. This is well shown by the study of the epidemics at Dublin as recorded by Grimshaw and Moore;[5] the epidemic of Brussels as described by Barella;[6] the epidemic of Toulouse as given by Bonnemaison;[7] the epidemic of Florence, of which Banti[8] is historian. Influenza prevailed in Toulouse in 1875, typhoid fever in Dublin in 1875, in Brussels in 1877, and in Florence in 1878. How, in

[1] Ritter, Deutsche Arch. f. Klin. Med., 1880, XXV.
[2] Muller, Endemische Pneumonie. Deutsche Arch. f. Klin. Med., 1877, XXI.
[3] Bielenski, Cité par Mendelsohn. Zeitsch. f. Klin. Med., 1883, VII.
[4] Couldrey, The Lancet, Nov., 1878; Herr, Verhandlungen des naturhistorichen Verein der Heilkunde, 1872; Heistch, Inaug. Dissert, Leipsig, 1882; Giornelli, Il Morgagni, 1877.
[5] Grimshaw & Moore, Irish Hosp. Gaz., 1875.
[6] Bull. Acad. de Méd. de Belgique, Feb., 1877.
[7] Bonnemaison, Union Méd., 1875.
[8] Banti, Arch. Gén. de Méd., 1880.

these different epidemics, are we to separate pneumonia from the predominating disease and assign its exact part to each? Hence it is, as we shall soon see, that the observers have put forth the most discordant opinions concerning the true nature of these epidemics. We shall do well, then, not to commit ourselves wholly to any theory as to the nosological classification of these epidemic pneumonias of the great cities till further enlightened as to the nature and causation; while at the same time it does not appear to us so difficult to determine the true character of the little epidemics of village or of family.

CHAPTER XIII.

UNITY OF PNEUMONIA.

Etiological Unity of Sporadic Pneumonia and of Epidemic Pneumonia.

The old idea of the frankly inflammatory nature of fibrinous pneumonia is so rooted by tradition in the medical mind, that to explain the facts which we have just related, no other means has been found than the admission of the existence of two distinct kinds of pneumonia, the one frank, due almost always to the action of cold; the other infectious, caused by a specific morbid agent. According to certain authorities, this second variety is subdivided into pneumonias due to a miasm peculiar in its relation to the lungs, and pneumonias due to the primary, or predominant, fixation in the lungs of a virus habitually affecting a different localization, such as that of typhus or typhoid fever.

As far as this last subdivision is concerned, it is necessary to distinguish the fact itself, and its theoretical application. We shall have to discuss the typhoid pneumonias when we come to speak of the relations between pneumonia and typhoid fever; we may from henceforth admit the reality of such pneumonias, but it is not possible to explain all the epidemic pneumonias by the above named hypothesis. Hence it is that though this explanation has been invoked by writers such as Barella, yet he does not extend it absolutely to all the miasmatic pneumonias. He thinks only that certain Summer pneumonias must frequently be the result of a typhous-poison. But the observations of Barella were made in a great city, and it is the epidemic of Brussels which inspired him with this hypothesis. His opinion is, moreover, founded chiefly on comparison of the tables of mortality from typhoid fever and pneumonia in the city of Brussels. We shall refrain from entering into a discussion of these statistics. As we have said, the complexity of causes in the case of pneumonic epidemics of the cities is too great to allow us to deduce as yet any very satisfactory conclusions as to the true etiology of the disease from the study of these epidemics.

Bonnemaison, writing concerning the epidemic of Toulouse, brings in the ancient notion of malignity. There is no essential difference between frank pneumonia and epidemic pneumonia. Only, at certain epochs, in certain conditions there is developed a special medical constitution imposing on all the diseases of the period a character of malignity. This is what he calls the septicæmic constitution, which creates the malignant pneumonias, as it causes the gravity of erysipelas and of puerperal or typhoid fever observed at the same epoch. Under certain ill understood influences, pneumonia, like any other ordinary affection which is benign and sporadic, may become epidemic, and, by the fact of a poison originating in the organism, and hence *auto-septicæmic* (or *hetero-septicæmic* in case

the poison comes from without), may acquire the characters of virulence and contagiousness.

Banti, Grimshaw and Moore make a clear distinction between the pneumonias which they have observed and ordinary frank pneumonia. The epidemic of Florence began in the autumn of 1877 and did not cease till the spring of 1878. It had been preceded by an epidemic of typhoid fever. But despite this circumstance, Banti refuses with Barella to see in these facts cases of pneumo-typhus, by reason of the absence of all lesion of Peyer's patches. He believes that these pneumonias were due to a special miasm, different from the typhoid miasm. This is also the opinion of Grimshaw and Moore, who, notwithstanding the existence of an epidemic of typhoid fever, attribute the disease to a particular miasmatic influence, and give to this form of pneumonia the name of *pythogenic* pneumonia (pneumonia engendered by putrefaction). "While ordinary pneumonia," they say, "prevails during seasons of protracted cold weather accompanied by severe winds and extreme variations of temperature, pythogenic pneumonia attains its maximum in warm seasons, characterized by dryness, a burning sun and rapid evaporation."

Rodman and Kuhn assign the principal rôle in the production of the epidemics which they have observed, to overcrowding, bad hygiene, and filthiness of the prisons. There is, according to Kuhn, a direct ratio between overcrowding of the rooms and dormitories, and the prevalence of the epidemic. "At the Kentucky prison hospital," says Rodman, "the hygienic conditions were deplorable. The fæcal matters were allowed by the prisoners to accumulate in receptacles in their cells, and you may imagine the intolerable odor emanating from this source. This stench was worse and worse as you went from floor to floor of the jail, and when you arrived in the upper story the fetor was horrible beyond description. I have seen persons scarcely able to refrain from vomiting on entering one of the cells of the upper story. One half of the cases and of the deaths occurred among prisoners occupying the cells near the roof."

No one can deny the causal agency of overcrowding in the development of epidemics; but it is an etiological factor common to all epidemic diseases. Overcrowding gives wide extension to the epidemic affection, but cannot be regarded as anything more than a predisposing cause, whether the epidemic take the name of typhus or typhoid fever, dysentery, pneumonia, or puerperal fever. From the fact that under the influence of conditions such as are described by Rodman, pneumonia took on a development somewhat different from its ordinary course, one is not authorized on this account alone to make of that form of pneumonia a distinct species. Rodman admits, besides, that at the time when this epidemic prevailed, there were present at the prison hospital numerous cases of frank pneumonia. Moreover, while making due account of the filthiness of the localities invaded, of the bad hygiene of the inmates, it will not do to exaggerate the value of these etiological conditions. They are in no sense necessary to the development of epidemic pneumonia, for in numerous family epidemics it has been especially noticeable that the patients were living in the very best conditions of aëration and of hygiene.

On what grounds, then, can any one assign a place apart, *i.e.* the right and title of a distinct species, to epidemic pneumonia? Is it on the typhoid aspect of the disease, the march and gravity of the epidemic? But every infectious disease may be benign or fatal without for that reason

changing its nature. Do nosologists constitute different species of typhoid fever because one epidemic is mild and another is of ataxo-adynamic nature? Do they make a specific differentiation between diphtheria which is limited to the tonsils, and that toxic form which overwhelms and kills the patient in three or four days? Adynamia and ataxia cannot of themselves constitute a species. Moreover, if epidemic pneumonia sometimes takes on a character of great malignity, so as out of five patients to kill five, as in Patchett's local epidemic, it is far from being always mortal, if indeed it be generally more grave than sporadic pneumonia. In the epidemics which we have mentioned, by the side of cases of the greatest gravity there were also observed cases as light as those of the most ordinary form of sporadic pneumonia. The highest mortality was that of the second epidemic of Frankfort, namely 25 per cent; at Moringen there were sixteen deaths out of eighty-three patients in the first epidemic, and eight out of fifty-eight in the second; in the first epidemic of Frankfort the mortality did not exceed 8 per cent. But what is the average mortality of simple frank pneumonia in adults from thirty to forty years old? Twenty per cent. But it is a striiking fact that certain village and family epidemics have been remarkable for their benignity. In that recorded by Parkert, there were only two deaths out of forty-two patients; in the family epidemic of which Bielenski gives an account, in nine cases there were nine recoveries.

Can any one say that the march of the disease is different? Doubtless it is natural to point out the frequency of prodromes, the more tardy appearance of physical signs, the renal, pleuritic, pericardial complications, etc. But all these characters may be met with in sporadic pneumonia and in epidemic pneumonia, and yet we shall not fail to find that feature which is the most essential attribute of frank pneumonia, namely its cyclical evolution. In Muller's epidemic, where five members of the same family were successively attacked, the defervescence took place, as it ordinarily does, on the fifth, seventh, and ninth days.

There is, then, nothing in the symptomatology, the march, or the gravity of the disease which authorizes an absolute separation between the two forms of pneumonia. Etiologically, pneumonia is rare, whether it be sporadic or epidemic. It is an infectious endemic disease, which in certain conditions may take on the epidemic character, like all the infectious diseases. There are differences of degree in its gravity, differences of aspect in its clinical evolution; but these differences are the same in the sporadic as in the epidemic state. The disease may remain limited to the lung or invade the neighboring organs and extend more or less to the rest of the organism: there is a *localized* pneumonia and an *infectant* pneumonia, but there is not a *frank* pneumonia and an *infectious* pneumonia.

There is one question, nevertheless, which may be asked, to wit, whether the parasite is the same in all the epidemics of which we have account, or whether with the ordinary parasite there may not be conjoined in certain cases other microphytes? This is an affair for future researches to decide. For the present, and till further enlightened, we may affirm that fibrinous pneumonia, sporadic or epidemic, has always for its essential cause the ellipsoid micrococcus.

Contagiousness.

Pneumonia being demonstrated to be an infectious and parasitic disease, the first of the etiological conditions to examine is that of its contagiousness, which Grisolle declared to be an unfounded hypothesis. The

contagiousness of pneumonia is, however, no longer an untenable tenet, but facts render it more and more probable. Authorities nevertheless still remained divided on this question. The greater part of medical observers who have witnessed epidemics of little extent, and in particular family epidemics, declare themselves convinced of the contagiousness of the disease. Struck with the mode of development of these pneumonias, attacking in succession several members of the same family, they cannot help seeing there a proof of the contagious nature of the affection.

One might invoke in support of the hypothesis of contagiousness the observations which we have cited of Wynter Blyth, of Hardwick, of Bonnemaison, of Daly, of Patchett, of Müller; sometimes it was individuals living together in the same house in continual contact with the patients who took the disease; sometimes, and the demonstration seems still more conclusive, persons in perfect health, dwelling in a distant quarter of the city or in a neighboring city, came to see a pneumonic patient, and were smitten in their turn after a sojourn more or less long in the morbid locality. Thorensen says that he saw five persons attacked with pneumonia, after having nursed some pneumonic patients in the English epidemic of Afghanistan. Costello relates that in the hospital where the pneumonic soldiers were treated, patients affected with different diseases contracted pneumonia,—"And even the hospital servants, including a hospital assistant," he adds, "took the disease, apparently, from the pneumonic patients whom they were at the time attending." In the first epidemic of Moringen jail, Kuhn likewise observed cases where nurses, and even patients with chronic diseases, and sleeping in the same ward as the pneumonic patients, took the contagion. Certain of the prison officers living in the city, outside of the prison, also got the disease from contact with the prisoners, and some of them gave it to members of their families. Kuhn relates the following fact, which concerns him directly, and which constitutes a most important testimony in favor of the contagiousness of pneumonia:—"I should add that I myself at the beginning of March suffered for several days with a febrile attack accompanied by swelling of the spleen. At the same time my coachman, who cleansed my garments, was also ill, though not seriously; then it was the domestic's turn, who during the illness of the coachman, took his place in the cleansing of the garments. Finally, almost at the same time, the disease attacked my little girl, who, notwithstanding my remonstrance, often ran out to meet me and embraced me when I returned from the prison before I had changed my clothes. In the case of these two last subjects, the disease announced itself by certain vague prodromes, then by a violent fever, accompanied with convulsions in the child, with delirium and obstinate vomiting in the servant maid. Towards the fifth or sixth day we noted in spots in the lungs, foci of hepatization, which developed one after the other as in migratory pneumonia. This is not all: a young girl who came to take the place of the sick domestic, was smitten in her turn. Finally, as a last episode, the domestic was sent during convalescence to her parents in a neighboring village three hours' ride from the city; she carried with her several of the woolen garments which had served her during her sickness. Eight days after her arrival, her sister, who slept with her, fell sick and presented all the symptoms of infectious pneumonia." It is very difficult, unless we change the sense of the word contagiousness, not to see in these facts examples of the possible transmission of pneumonia by contagion. The proofs are the same as those

on which rests belief in the contagiousness of diphtheria, of typhoid fever, of cholera, of small-pox, etc. When we find ourselves in presence of facts so clear and striking, have we the right to assume simple coincidence, solely because the facts are in contradiction with the traditional teaching? Setting aside all theories, what difference is there between the familiar instances of diphtheria destroying a whole family in several days, and the observation, for example, of Patchett, where an entire family of five members succumbed in a fortnight to the fatal ravages of pneumonia?

A certain number of observers, nevertheless, affirm that they have not noticed instances of contagiuosness in the epidemics which they have described. In the epidemic of Noyers, Torchet did not obtain any facts favorable to this view, although the subject particularly attracted his attention. He says that among the persons who had daily relations with the patients, such as the nurses and attending physicians, although the latter did not hesitate fearlessly to come in contact with the sick and to auscultate them without stethoscope, the ear being immediately applied over their sweaty linen, yet no one was attacked by the prevailing disease.

Rodman, Grimshaw and Moore, also, do not believe in the doctrine of contagiousness; they have never seen cases resulting from contact, mediate or immediate, with patients affected with pneumonia. Kuhn himself declares that he did not observe a single case of contagion in the second epidemic of the Moringen prison.

In these conditions the simultaneous or successive development of a great number of cases of pneumonia is explicable by the existence of the same agent of infection, smiting one after another, individuals living in the same locality. We may then conclude, that we are not yet in possession of sufficiently complete data to warrant us in the assertion that the contagiousness of pneumonia is proved. The cases cited above, however, authorize us in regarding this view as one of increasing probability.

Atmospheric Conditions.

Among the adjuvant conditions which may favor the development of pneumonia, the most interesting to study are the variations of the atmosphere, and in particular the part played by the different seasons of the year.

Seasons.—We do not attribute to variations of seasons, the importance which has been assigned to them by Jurgensen and those who have followed him in his way of demonstrating the infectious nature of the disease. If there were no other and better arguments with which to substantiate this theory, it would fall to the ground. Besides, the statistics appealed to as demonstrative, are absolutely puerile, and prove nothing. To one set of statistics, it is always easy to oppose another set. If, for instance, Jurgensen's tables establish differences, as far as the point of view of seasons is concerned, between the curve of pneumonia and that of bronchitis, those of Buchan and Mitchell, based on the mortality of London during thirty years, from 1845 to 1875, strongly demonstrate the contrary—to wit, that the curve of mortality from pneumonia is essentially the same as that of mortality from bronchitis. "This curve," say these two writers, "is constant from year to year, having its maximum in the cold seasons and its minimum in the warm seasons." But what value should be attached to such observations and such statistics? How can one reasonably think of making a comparison between bronchitis and pneumonia by basing oneself on their respective mortality? Do we often witness attacks of bronchitis

à frigore, unconnected with and apart from suffocative catarrh, capable of causing the death of the patient? Is it not evident that under the denomination of bronchitis, employed in mortuary statistics, is confounded a multitude of different affections, from pulmonary tuberculosis to diseases of the heart? On the other hand, do statistics make any difference between primary and secondary pneumonias? How, then, in the midst of this confusion are we to distinguish *things* from *words?* Is it not better to leave altogether to one side considerations so vulnerable to criticism?

It, however, matters little, from the point of view of the nature of pneumonia, whether this disease follows or not the curve of frequency of bronchitis. Supposing even that pneumonia were more frequent in the cold season than in spring or summer, what could this fact prove against the theory of its parasitic and infectious nature? If there be any disease whose infectiousness and specificity are no longer questionable, it is assuredly diphtheria. But if you will take the trouble to look over the hospital statistics, published every quarter by Besnier in the Bulletins de la Société des Hôpitaux of Paris, you will see that every year the mortality from diphtheria attains its maximum during the winter months.

Let us, then, abandon these idle discussions, and content ourselves with the simple inquiry as to the influence of the seasons on the frequency of pneumonia. On this point all observers are pretty well agreed in our day, in rejecting the old notion that pneumonia is a disease peculiar to the winter. Even Sydenham has said that the maximum of frequency is rather between spring-time and summer, and Van Swietan supports the same view. The statistics of Grisolle bearing on 553 cases, fully confirm the assertion of Sydenham, and we may regard these statistics as fairly representing the observation of each day. Of these 553 cases, 318 belonged to the months of February, March, April and May, and of these 318 cases March and April claimed 186. Pneumonia is, then, a disease of spring-time and not of winter. It prevails sporadically the whole year round, but it undergoes every year a veritable epidemic recrudescence, and this recrudescence occurs in the early part of the spring.

Climates, Winds, Barometric Pressure.—We shall not dwell on these divers conditions, concerning which there exist no data worthy of serious attention. We need only note that pneumonia has been observed about equally in all quarters of the globe, save in the equatorial and polar regions, where it is in fact unknown, or at least very infrequent.

Hygrometric States.—According to Jurgensen, the state of humidity of the atmosphere exercises a great influence on the development of the disease; injurious when the hygrometric condition is above the mean, favorable when it is below. Ellissen also affirms a certain relation between the frequency of cases of pneumonia and the quantity of moisture contained in the atmosphere. In fine, in a communication recently made to the International Medical Congress at Copenhagen (August, 1884), Fleudt declares that a single fact only stands out prominently as a result of the study of the relationship of pneumonia to atmospheric conditions, namely the influence of moisture in the air; the frequency of the disease being directly proportional to the curve representing the rainfall. This predominant importance of the hygrometric state agrees perfectly with the demonstration of the parasitic nature of pneumonia, and with the idea of a microphyte having the soil for its ordinary habitat. In the case of other infectious diseases, and diphtheria in particular, this remarkable influence of atmospheric humidity on recrudescences of the disease is of daily observation.

Putrid Miasms and Sewer-gas.—Vitiation of the air by putrid emanations has been alleged as one cause of pneumonia, being especially a factor in the causation of certain epidemics. It is thus that Banti explains the epidemic of Florence by the effluvia set free by the detritus accumulated by the sewers of the city in the bed of the Arno, which was nearly dry in a season of drought. In the account which he gives of an epidemic of pneumonia, Penkert remarks that the school building where the epidemic broke out faced the village cemetery, and that just back of the cemetery was a flat occupied by a little pond. To judge by the height of the water in the pond, the soil of the graveyard, which was quite porous, had been invaded by infiltration of the water, whence issued fermentation of the detritus buried there, and the setting free of infectious germs which were carried about by the wind. Now the northwest wind, which was prevailing at that time, swept only one house in that village, namely the school-house, and it was a fact that the rooms exposed to the blasts from the northwest furnished the first victims to the epidemic. Grimshaw and Moore—as the very term which they have given to the pneumonic epidemic which they observed implies, viz., *pythogenic*—assign an important part to putrid miasms set free from the soil by rapid evaporation, and they relate several cases of pneumonia in individuals whose unhealthy lodgings were directly exposed to the emanations of the sewers.

The English have dignified these pneumonias with a special name—*sewer-gas pneumonias*. One of these epidemics, where the influence of these putrid gases seems sufficiently evident, deserves mention.[1]

The observation concerns a school situated near the mouth of a sewer. On March 14th, 1874, the mouth of the sewer was opened, and a ventilator, protected by a charcoal filter, was put into it. March 20th the pressure of the gas in the sewer was suddenly augmented by reason of a high tide; the gas made irruption by way of the ventilator, and some of the domestics of the institution who slept in the chambers looking out on the street, complained of the bad odor. The next morning, one of the children who slept in a room situated right opposite the ventilator, was taken with an attack of grave pneumonia. During the day and the evening, two other pupils and two domestics were attacked with the same disease. One of the domestics died. That very day an order was given to remove the ventilator, and to close up the sewer, and from that time there was not another case of pneumonia. Whatever interest may be attached to these facts, they do not prove anything other than this, that the germs of pneumonia may be mingled with other germs of putrefaction, and that cultivated in a putrid medium they there possibly acquire a higher power of infection, a greater degree of virulence. These same sewer-gas emanations have been, in fact, often invoked to explain the development of epidemics of typhoid fever or cholera, and it is plainly evident that considered in themselves, and independently of the specific germ, they cannot be held accountable for the production of three diseases so unlike.

Other Favoring Conditions.

Chilling of the Body.—This, according to the older physicians, is the true cause of pneumonia. Pneumonia is a disease due to a chill; *frigus unica causa pneumonica*, such was the formula. There is scarcely

[1] Med. Times & Gaz., April, 1874. See also Legendre, Union Méd., Feb., 1883.

need of discussing such a proposition at the present day. If this view still claims a few partisans, it is owing to two reasons:—first to the power of the traditional notion; second, as Grisolle has already remarked, to the statement of the patient, in response to the usual interrogatory. If you simply ask the patient if before the attack he had a chill, he will almost always reply in the affirmative; but if you take the pains to examine into the affirmation by new questions, you will find out either that the patient understands by the chill the initial rigor, which is in fact characterized by a very marked sensation of cold, or that the chill goes back to a period so distant from the invasion of the disease that it is impossible to assign to it a causal connection; or it may be, lastly, a mere impression on the mind of the patient, who, unable to explain the occurrence of the disease in any other way, imagines himself to have contracted it by a chill, though he does not know where or when. The patient, in fact, reasons after the fashion of some physicians who assume the causal action of cold as indispensable, even when it is impossible for the patient to indicate the time, place, or circumstance where the chilling could have occurred to produce the disease. We must then suppose, they say, that the patients were inattentive to the sensations of cold which they must have experienced; a mode of reasoning peculiar to those who have a dogma to maintain, but hardly defensible in the light of clearer notions of etiology. In fact Andral and Chomel long ago showed the fallacy of this theory, dogmatically maintained by certain authorities. They recognized the fact that among individuals who are exposed to severe chilling while the body is in a state of perspiration, only the smallest fraction is attacked by inflammation of the lungs; and that often, on the other hand, pneumonia manifests itself without our being able to discover the intervention of any occasioning cause whatever. The exact statistics of Grisolle, the careful observations of recent times, have completed the overthrow of the theory of bodily chill. Out of 205 patients, interrogated from this special point of view, only 49 ascribed their pneumonia to exposure to cold. This is about the proportion indicated by Gerhardt, who out of 166 cases of pneumonia, found only 33 in which a previous chilling was recognized as the occasioning cause. The ratio noted by Ziemssen is still less; out of 106 cases of pneumonia he was able in only 10 instances to prove the previous existence of bodily chill. Griesinger has arrived at even a smaller proportion, viz., less than 2 per cent.; in 212 cases occurring at the Zurich clinic, only four times was there noted any immediate connection between exposure to cold and the inception of the disease. "For ten years," says Jurgensen, "I have not only caused to be enregistered at my clinic the cases in which the patients had a bodily chill, but I have taken pains to have the nature of such chill ascertained. I have thus arrived at the figure of 10 per cent.; but I should add that on careful investigation I have been compelled to lower the proportion to 4 per cent." At the last Medical Congress in Copenhagen, Flindt gave the proportion, as carefully estimated by him, at 8 per cent. "In 92 per cent. of all the cases," he adds, "the supposition of any kind of a chill had to be absolutely excluded." These figures suffice to do justice to the pathogenic rôle attributed to cold in the development of pneumonia. If chilling be the real cause of pneumonia, how explain the fact that in three fourths of the cases (the statistics of Grisolle, which give the highest proportion, alone being excepted) the disease may be produced without the intervention of this cause?

We do not, however, refuse to bodily chilling all pathogenic influence;

but in the development of pneumonia, as in that of other diseases, it acts only as an adjuvant, occasional, accidental, but by no means necessary cause. Jurgensen admits that in certain cases cold acts in provoking a catarrhal inflammation of the bronchi, which favors the development of germs introduced into the lungs. We are the rather inclined to think that the action of cold is more strictly of a massive order, *i.e.*, by disturbing the vascular and nervous equilibrium of the lungs it enfeebles the resistance offered by the pulmonary tissue to the invasion of the parasite foe, and this enfeeblement of cellular vitality realizes probably one of the conditions favorable to the germination of the microbe.

Traumatism.—In a few cases, of rather doubtful kind, where frank pneumonia, according to the statement of observers, has succeeded a contusion or traumatism of the thorax, the same explanation may be invoked. The pneumonic microbe was already an occupant of the pulmonary tissue ready to undergo development; the traumatism has only served to precipitate and favor its multiplication and its extension, by suddenly diminishing the normal resistance of the pulmonary textures.

In most cases, in fact, it is not frank pneumonia which is produced as the sequel of a traumatism. Whether it be an affair of contusions of the thorax, of fractures of the ribs, of penetrating wounds by sabre or fire-arms, of foreign bodies introduced by the bronchi, of irritating vapors inhaled, you will observe effusions of blood, purulent or gangrenous inflammations, broncho-pneumonias, but nothing which resembles the primary pneumonia of the elliptical coccus.[1]

Overwork and Overcrowding.—These two conditions intervene in the development of pneumonia in the same auxiliary manner as in all other infectious diseases. The one favors the multiplication of the morbigenous germ in the over-fatigued organism, the other the multiplication of cases of the disease in the overcrowded locality. They act also by stamping the symptomatic aggregate with a special aspect, which is characterized by the name of *typhoid* or *adynamic*. But neither the one condition nor the other is capable by itself of determining pneumonia.

Individual Conditions.

We include under this head the conditions of age, sex, constitution, race, profession, which claim but a brief attention.

Age.—*Primary* pneumonia is a disease of all ages. It is nevertheless more frequent in adult life and old age; children of first dentition are more exposed to broncho-pneumonia.

Sex.—Since Cœlius Aurelianus, authorities have been agreed in recognizing that pneumonia is more rare in females than in males. Attempts have been made to show this fact by statistics founded chiefly on hospital records. Is not this relative immunity of the female explicable simply by the circumstance that women consent less willingly than men to be taken to the hospital to be cared for there when sick? In opposition to the hospital returns, country physicians have observed the same frequency of the disease in both sexes. Thus it is that in the Grand Duchy of Bade, Ruef, out of 94 pneumonic patients treated by him in the space of three years, found 44 males and 50 females. In the Ain, Munaret, in 37 cases, noted 20 males and 17 females. According to Toulmou-

[1] See on this subject the thesis of Proust on Traumatic Pneumonia; Paris, 1883.

che, in the prison of Rennes the disease is as frequent in the women's division as in that of the men. Before seeking an explanation of the popular opinion, it would be well first to assure oneself of its truth.

Constitution.—We may say as much of the assertion of Hippocrates, who regards persons of robust constitution as more subject to pneumonia than individuals of weak constitution, and of that of Bouillaud, who considers the lymphatic or sanguino-lymphatic temperament as constituting a true predisposition to this disease. Here is worth noting, as being of interest in this connection, the opinion of Luzinski, based on a wide experience, that children born of parents affected with chronic affections of the chest are predisposed to pneumonia.

Race.—The English statistics cited by Grisolle, establish the fact that among the colonial troops, the negro soldiers are oftener attacked by pneumonia than the white soldiers. The facts observed by Rodman in the epidemic of Frankfort jail, seem to confirm these data. Although the proportion of white and of black convicts was nearly the same, the number of negro patients exceeded that of white patients by fifty, and out of 25 fatal cases, Rodman reckoned 24 negroes and only one white man.

Profession.—There is no satisfactory conclusion deducible from any figures collected, to show that pneumonia selects any one profession or occupation preferably to another. Once more we must not forget that these statistics are based on hospital observations. And when Chomel makes the proposition that the occupations which furnish the greatest contingent of pneumonic cases are those of carpenter, domestic, mason, it seems to us to prove only one thing, namely, that carpenters, domestics, and masons are oftener obliged to have recourse to the hospitals when sick than tailors, butchers or grocers.

State of Health or Disease.—Pneumonia may surprise the individual in perfect health. But every cause of general debilitation, every chronic disease of long duration, may be considered as an adjuvant condition. The frequency of pneumonic lesions in patients who succumb to an affection of long standing, is the proof of this. Without doubt a certain percentage of these lesions should be withdrawn from frank pneumonia, and assigned to the category of passive congestion from dorsal decubitus, or hypostatic pneumonia, or of pulmonary infarctions. But the larger part are manifestly red or gray fibrinous hepatizations,—identical with those of true pneumonia. Diabetes, albuminuria, cancer, often end in pneumonia. Grisolle notes inflammation of the lungs as an intercurrent disease in a seventh of the cases of chronic lesions of the liver, and in particular of cirrhosis. Out of 65 observations of diseases of the heart, the same pathologist found in 18 cases, *i.e.* more than a fourth, a pneumonia supervening as final lesion. Chambers has noted a proportion of ninety-six cases of hepatization of the lungs in a hundred and five autopsies of cardiac patients. We often hear remarked, moreover, the frequency of pneumonia in the apoplectic and also in old hemiplegics, and in almost all these cases, according to Rosenbach, the disease is on the paralyzed side. It is the same, according to alienists, in the case of the demented. A tenth, according to Bayli, a fifth, according to Calmel and Bouchut, a third, according to Lawrence, succumb to an intercurrent pneumonia. Lastly, in dangerous surgical operations, in compound fractures, extensive wounds and burns, etc., it is pneumonia which most often comes in to terminate the scene, and this independently of all purulent infection. Out of forty-

one cases of death from various surgical affections, Erichsen observed in twenty-three pneumonia in the first or second stage.

In each and all of these cases, one may say boldly that the parasite of pneumonia, grafted on an enfeebled organism, invades the lungs, just as the *oïdium albicans* of thrush under similar conditions invades the lingual and buccal mucosa.

CHAPTER XIV.

SECONDARY PNEUMONIA.

Specific Causes.

We understand by secondary pneumonias, as we have before said, pneumonias which develop in the course of different infectious diseases, generally, in all probability, under the influence of the specific agent peculiar to each of these diseases. We say generally, because it is certain that fibrinous lobar pneumonias corresponding exactly to the anatomical type of primary pneumonia, are observed in the course of influenza and typhoid fever. These pneumonias can only be explained in one of two ways: either the microbe of influenza or of typhoid fever may of itself produce a pneumonia similar to that of the elliptical microbe, or else there is secondary penetration and adjunction of this micrococcus to the specific agent of the primary disease; however this may be, in the immense majority of cases the lesions are those of lobular pneumonia or broncho-pneumonia.

Measles.—Measles is regarded by all authorities as one of the most common causes of secondary pneumonia in the child. According to the stastistics of Barrié, Damaschino, Roger, and Cadet de Gassicourt, measles occasions more than a fourth of the cases of broncho-pneumonia. This frequency varies nevertheless according to certain conditions, even in the child. Some epidemics are remarkable by the number of pulmonary complications. From this point of view, hospital epidemics of measles have a special gravity. In the infant wards, death by pneumonia is the rule in the case of infants under two years of age. In the adult, on the contrary, rubeolic pneumonia is, as a general thing, exceptional. Nevertheless in divers epidemics observed in adults, the frequency and gravity of the broncho-pneumonias have been the same as in the infant. As the rubeolic poison manifests its action from the first on the respiratory mucous membrane, the specific nature of these broncho-pneumonias seems unquestionable. The precocious development of the pulmonary lesions is still another evidence of the specificity. The direct and experimental proof has, however, not yet been made. We do not know the microbe of measles, and the micrococcus of Bobés, found in the exudation of morbillous broncho-pneumonia, differs in nothing from the micrococcus that is found in all bronchial products.

Diphtheria.—Diphtheria has the second place among the causes of the secondary pneumonias; but here the pathogeny of the lesions is evidently not always the same. Nodules of broncho-pneumonia may in fact be observed; 1. In pharyngeal diphtheria, but especially and perhaps only in the toxic form; 2. In laryngo-bronchial diphtheria; 3. After tracheotomy; 4. In cases of paralysis of the muscles of the pharynx and larynx. The specific nature is hardly evident except in the second group,

when the diphtheritic inflammation is propagated to the bronchial terminations. But doubt is permissible with respect to the three other categories; one may even go so far as to say that in the case of paralysis, the broncho-pneumonia is surely of mechanical origin, and that it is due to the penetration of buccal liquids or of alimentary particles into the lower bronchi, as in the experiments made on animals in which section has been made of the inferior laryngeal nerves. As for the broncho-pneumonias which develop as a sequel of tracheotomy, the two causes, specific and mechanical, probably act together. As for those which are observed in diphtheria localized in the pharynx, it is very likely that the mechanical influence has the principal part. The nodules of broncho-pneumonia are in fact seen almost exclusively in the toxic form. But in such cases the difficulty of deglutition is extreme; the irritant liquids stagnate in the mouth; the situation is the same as in cases of noma or cancer of the tongue; and we can easily understand the ready penetration into the air passages of irritant agents of all kinds, specific or not, accumulated in the fauces and pharynx.

Influenza.—In the case of influenza, as well as of measles, the frequency of pneumonic complications varies with the epidemics. The proportion observed by Landau is 26 per cent.; that of Copland, 22 per cent.; that of Lepelletier is 13 per cent. Biermer in a general way indicates only from 5 to 10 per cent. The pneumonias of influenza are especially formidable in the case of old people. The grave form habitually observed is sub-acute broncho-pneumonia with suffocative catarrh. In these cases the direct rôle of the epidemic influenzal agent is evident. But there exist other facts, where are noted at the autopsy the lesions of fibrinous pneumonia with an exudation of the same nature in the bronchi. We have already said that one may properly ask if, in such cases, the morbid principle of the influenza is alone concerned, or if it may not be associated with the parasite of primary pneumonia.

Pertussis.—In whooping cough, lobular pneumonia is certainly rarer than in measles and diphtheria. Its frequency nevertheless represents a fifth of the total number of cases of broncho-pneumonia observed in children's hospitals, according to Roger and Cadet de Gassicourt. It is especially liable to occur in pertussoid children under three years of age. Exceptional in the first week, this complication appears during the second or third week of the period of paroxysmal cough. According to Damaschino, the broncho-pneumonia of whooping cough localizes itself frequently in one lung, and even in one lobe.

Small-pox and Scarlatina.—Variola and scarlet fever are rarely causes of pneumonia. There are nevertheless exceptions to this rule, and in the case of variola especially, you will sometimes find at the autopsy broncho-pneumonic lesions. Jouffroy has observed them in the adult, in fully one half the cases which he has examined.

Erysipelas.—The relations of pneumonia to erysipelas will be studied further on; we shall have occasion to speak of what it will be convenient for the sake of precision to call *erysipelatous pneumonia*. From an etiological standpoint, the rôle of erysipelas of the face in the development of certain inflammations of the lungs, cannot be denied; the propagation of the specific phlegmasia by the intermedium of the mucous membranes of the pharnyx, larnyx and trachea is demonstrated by the facts of Gubler, Labbé, Strauss, Stackler, Damaschino, etc.

Erythema Nodosum.—Erythema nodosum, considered as a

specific fever, may be complicated with pneumonia, as the examples cited by Trousseau, Rondlot and Talamon attest. This nodose broncho-pneumonia ought nevertheless to be regarded as rare; it is more generally pleurisy which is observed in these cases.

Acute Articular Rheumatism.—The physicians of a former epoch singularly exaggerated the frequency of pneumonia in articular rheumatism. Barthez goes so far as to say that of all rheumatismal inflammations of the viscera, pneumonia is the most frequent. There is here evidently a confusion of pneumonia with pleurisy, which the older physicians do not distinguish from pneumonia; possibly also with simple pleurodynia. Nevertheless, while making all due allowance for the part played by congestions and emboli, which are under the dependence of grave rheumatismal endocarditis, and while recognizing the rareness of true inflammation of the lungs of rheumatic origin, the reality of this cause in a certain number of carefully observed facts must be admitted.

Typhoid Fever.—As in the case of erysipelas, there will be occasion farther on to discuss the relations of typhoid fever and pneumonia. Setting one side, for the present, the question of pneumo-typhus, we need only say that the constancy of the typhoid determinations to the bronchi, joined to the influence of dorsal decubitus and enfeeblement of the heart's action, sufficiently explains the frequency of hypostatic pneumonia and the nodules of broncho-pneumonia found at the autopsy of subjects dead from intestinal typhus.

Intermittent Fever.—The localization of the marsh miasm in the lungs gives rise to a special form of pneumonia described under the name of intermittent pneumonia.

Pernicious Pneumonia is the grave form of this variety.

Irritant Causes.

Gangrene of the Mouth. Cancer of the Tongue.—The various secondary pneumonias which we have just enumerated may be explained by the action of a specific cause in the lungs. The broncho-pneumonias which are observed in noma, in subjects affected with lingual cancroid, can be due only to a mechanical cause. They have the same etiology as the experimental broncho-pneumonias of animals, being the result of penetration into the bronchioles of irritant matters accumulated in the buccal cavity and fauces, and that have found their way into the air passages by reason of the impediment to deglutition.

Cachectic States.—We may admit a like etiology for the broncho-pneumonias which so often terminate the life of cachectic subjects. In children afflicted with chronic diarrhœa, in the rachitic, in catarrhal old men, the feebleness of the expiratory forces, the paresis of the bronchial muscles, favor stagnation of the bronchial products in the small bronchi; moreover the want of resistance of the organism facilitates the irritant action on the pulmonary tissue of the germs of inflammation contained in the accumulated mucosites, and consequently the formation of nodules of broncho-pneumonia.

CHAPTER XV.

PATHOLOGICAL ANATOMY.

The lesions of the acute pneumonias correspond to two quite distinct processes; the one appertains properly to primary pneumonia, and results in the fibrinous infiltration of an entire lobe; this is fibrinous lobar pneumonia; the other is characterized by the lobular localization and dissemination of the lesions; this is lobular pneumonia or broncho-pneumonia.

Anatomical Characters of Fibrinous Lobar Pneumonia.

Since the time of Laennec pathologists have recognized three aspects or periods in lobar fibrinous inflammation of the lungs. The first period is characterized by a special congestive state, which Bayle has designated pulmonary *engorgement*, the second period is that of fibrinous infiltration called red hepatization, the third is the period of gray hepatization.

Engorgement.—According to Stokes and Walshe, the first stage of pneumonia is not really constituted by the engorgement, but by a special state of dryness, of firmness, and of arterial injection of the tissue; the lung presents a bright scarlet hue, without exudation of any sort, serous or sanguineous, in the alveoli. Stokes says that he has noted this appearance of the pulmonary parenchyma in the vicinity of pneumonic foci which have reached the stage of engorgement or hepatization; he agrees, however, it is rarely possible to witness this.

The engorgement is, then, the first alteration plainly appreciable. The lung presents to the naked eye a violaceous or wine-lees color; it is heavier and offers a greater resistance than normally. When pressed between the fingers, it still crepitates, but you feel that it is more compact than ordinarily, as if gorged with liquid. It keeps the impression of the finger almost like an œdematous member. On section, the reddish tissue lets flow a great quantity of turbid and frothy sero-sanguinolent liquid.

Is this aspect characteristic, and does it enable us to differentiate the first stage of pneumonia from simple passive or hypostatic congestion? Authorities have pretended that the increased friability of the tissue under pressure of the finger belongs only to an engorgement frankly inflammatory; but this friability exists independently of every process of phlegmasia, and one may truly say with Bouillaud and Grisolle that it is difficult and even impossible in many cases, if you do not observe several hepatized portions, to determine from the simple cadaveric inspection, if the engorgement is really inflammatory or simply mechanical.

If the gross macroscopic lesions are insufficient, we are obliged to say as much of the microscopic lesions. The microscope shows only varicose dilatation and engorgement of the capillaries of the alveoli and bronchioles in the congested parts. The alveoli contain red globules, a few leucocytes and

large granular cells enclosing one or several nuclei. These large cells are the cells of alveolar endothelium modified by the pneumonic process; you will see here and there one of them adhering in certain points to the walls of the alveolus. There is no difference in this respect between what one will observe in the first stage of ordinary pneumonia, and what is observed in hypostatic congestion, or in splenization, or what authorities have called catarrhal pneumonia.

Red Hepatization.—In the second stage of pneumonia the lung presents the appearance of liver. It is of a dark brownish-red color, although the hue is not uniform, in places being violet-red, in others scarlet. On the surface, are seen more clearly than in the normal state, the septa which mark the separation into lobules, while the bronchial tubes and blood-vessels are still obvious to examination. There is no longer crepitation under pressure, and the tissue is impermeable to insufflation. Thrown into water, a fragment of hepatized lung sinks to the bottom like a piece of liver or spleen. Upon section, it scarcely exudes any liquid from the cut surface, but on scraping it with a knife, you obtain a very thick reddish serosity, in which are distinguished minute granules or cylinders of a whitish substance, which are shown by the microscope to be fibrinous moulds of the alveoli and bronchioles. The surface of the section offers a characteristic aspect, which belongs only to fibrinous pneumonia and to the hæmoptoic infarctus. It is granular, as if studded with little roundish globular masses, which it is easy to recognize as the pulmonary vesicles themselves, transformed into solid grains by the concrete fibrinous exudation which stuffs their cavities.

Moreover the pulmonary tissue, despite the induration, has become more friable; it is readily torn, allows itself to be easily penetrated by the finger; and this it is that led Andral to prefer for this stage the name of red softening (*ramollissement rouge*) to that of red hepatization.

In fine the lung, having become in appearance only more voluminous according to Andral and Laennec, has in reality acquired a weight which is much greater than the normal, and this is the point of chief importance. The mean weight of the healthy lung being 680 grammes in man and 512 in woman, you will see it double and even treble in weight, especially in pneumonias which invade a whole lung. Grisolle says that he noted in one case a weight of 2500 grammes. Lépine observed in a female patient, a hepatized lung weighing 1700 grammes. This augmentation of weight results both from the accumulation of blood in the vessels, and the fibrinous exudation in the alveoli, the latter being the principal factor. According to the researches of Lépine, confirmed by those of Hamburger, the average weight of the pneumonic exudation is 600 grammes; pathologists have always found it above 400 grammes, and often it exceeds 1000 or even 1200 grammes.

Histologically, the process of the second stage is characterized by the coagulation of the fibrine which is effused into the alveoli, along with an abundance of red globules and leucocytes. On section the fibrinous exudation is seen in the form of a fine fibrillary network imbedding in its meshes the red and white globules, large round cells and endothelial cells more or less swollen, deformed and granular. This exudation fills not only the alveoli but also the cavities of the terminal bronchi.

It is in the midst of this fibrinous network, and in the granular cells, that is found the micrococcus, the cause of the lesions. It is very difficult if not impossible to perceive these micrococci in a preparation of the

lung hardened in alcohol; if you wish to study them to advantage *in situ* you must make use of frozen sections of fresh pieces. The simplest way is to scrape off a little of the exudation, spread it on the glass slide and allow it to dry; the preparation may be stained by Friedländer's process before described. Cornil prefers the following method: Dip the glass slide into a solution of gentian violet and aniline water; after keeping it there one hour, remove it and decolorize the preparation in a five per cent. solution of iodide of potassium. At the end of several minutes, re-stain it with éosine, then wash it in distilled water, then in alcohol, and mount in Canada balsam. On the red background the microbes appear stained violet.

In the midst of numerous round cocci, which are visible there as in every inflammatory product, you distinguish the elliptical micrococcus, like grains of wheat, isolated, two by two, or in chains of three or four. Very often these round or ovoid cocci are surrounded by a clear space more or less sharply defined, also roundish or elongated; it is this clear space, which has nothing characteristic about it as pertaining to the coccus of pneumonia, which has been regarded and described by Friedländer as a capsule peculiar to the microphyte. Often, moreover, when the exudation is examined a little too long after death, you see in the midst of these round and elliptical cocci, long bacteria, isolated, or in chains of two or three, which belong to the process of putrefaction.

Gray Hepatization.—This is the third stage described by Laennec. The consistency of the lung has diminished; the tissue is more friable, the finger in pitting forms cavities which, if one is not careful, might be mistaken for abscesses. On section there exudes a great quantity of a grayish-red liquid, always mingled with fibrinous particles, but formed chiefly of pus globules. The gray coloration is the most advanced stage of the lesion; the tissue may present a yellow color; Rindfleisch has given to this aspect the name of *yellow hepatization*. It is this appearance which Laennec had especially in view in his description: "The entire lung takes on uniformly a straw-yellow or citron-yellow color, and exudes more or less abundantly from cut surfaces a yellowish, opaque, viscous and evidently purulent matter of an insipid odor, though not so disagreeable as the pus from an external wound." The pus, according to him, takes on the grayish color when the lung contains a great quantity of black pigmentary matter, as is very common in adults and old people.

Under the microscope the fibrinous reticulum has in great part disappeared. You now observe nothing but numerous lymphoid cells, filled with fatty granules and disseminated through a more or less abundant granular matter.

Anatomical Modifications of the Exudations.—Without losing the characteristics of lobar pneumonia, the exudation may present histologically certain modifications. These modifications pertain especially to the quantity of coagulable fibrine. The proportion of this substance may be so trifling as, according to Schutzenberger, to constitute what may be described as a serous pneumonia, the liquid which fills the alveoli resembling rather the serosity of œdema than inflammatory lymph. Schutzenberger also describes a hæmatoid pneumonia, in which the alveolar exudation is formed of almost pure blood, *i.e.*, of red globules heaped together. In fine, in some cases observed by Ranvier, during the siege of Paris, and cited by Lépine, leucocytes predominated from the very first

day in the exudation. In some patients who died from pneumonia on the third day, but little fibrine, or none at all, was observed in the alveoli, which were almost exclusively filled by pus globules.

Banti, in the epidemic of pneumonia at Florence, has also indicated as a peculiarity of the pulmonary exudation, its poorness in fibrine and its richness in red globules. In the first stage especially, he says, the hæmorrhagic exudation exceeded everything which is ordinarily observed; the alveoli were stuffed with red globules mingled with leucocytes imbedded in the midst of a few fibrine filaments. The small bronchi and inter-alveolar connective tissue were also infiltrated and stuffed with blood globules, nevertheless the alveolar epithelium was intact and did not undergo change till in the second stage. The cells of the alveolar walls were then swollen, their nuclei were undergoing proliferation, and a certain number were found in the midst of the alveolar exudation. There was at the same time a certain amount of cellular proliferation, in the interstitial, inter-alveolar and inter-lobular tissue. Although Banti relies on these modifications of the exudation in making of the Florence epidemic a species distinct from fibrinous pneumonia, there is not really any absolute difference warranting such a conclusion. It is well, however, to make a record of this hæmorrhagic aspect of the exudation, which affiliates the pneumonias observed by Banti with the hæmatoid pneumonia of Schutzenberger; but we cannot regard it as characterizing epidemic pneumonias, for nothing like it has been noted in the other epidemics.

Extent of the Lesions.—The extent of the lesions is very variable. In general at the autopsy nearly the whole of one lung is found hepatized; sometimes only one lobe is affected, in other cases there is invasion of both lungs. According to the statistics of Vienna, which pertain to 10,000 cases of pneumonia observed from 1866 to 1876, the frequency of the localization in the right lung alone is in the ratio of 50 per cent.; in the left lung alone, 35 per cent.; in 15 per cent. only of the cases was double pneumonia observed. Ordinarily the three stages of the inflammation are found associated in the same lung, and divide it into as many zones insensibly shading into each other. Sometimes it is engorgement which predominates, and one observes here and there parts more resistant, more friable and granular, sinking in water. Grisolle declares that he has never, however short may have been the course of the disease, met with engorgement as the sole lesion, without accompanying red hepatization more or less extensive. In other cases, but more rarely, the whole of one lung presents the lesions of gray hepatization; there is then found habitually in the other lung commencing red hepatization.

Lesions of the Pleura and Bronchi.

Fibrinous inflammation of the pleura almost constantly accompanies primary pneumonia. Out of thirty-five cases in which the state of the serous membrane was noted, Grisolle found it intact but twice. The exudation is the same as that of the alveoli; the same fibrinous network imbedding leucocytes and proliferating and granular epithelial cells. The false membranes form ordinarily only a thin pellicle over the hepatized regions; they may nevertheless attain to some thickness; the inflammation may gain the costal pleura, and a sero-fibrinous exudation be poured out.

As for the state of the bronchi, according to certain pathologists, and particularly Andral, bronchitis constantly co-exists with pneumonia;

Grisolle, on the other hand, refuses to see in the reddening of the bronchial mucosa anything but simple active or passive congestion, not an inflammatory condition. If we take account only of the state of the large bronchi, we must admit that bronchitis is often wanting, and not merely often, but generally. But it cannot be denied that the bronchioles, the capillary bronchi, take part constantly in the process which has its principal seat in the alveoli. The existence of little fibrinous moulds reproducing exactly the form of these bronchioles, is in fact the rule; these may be detected during life by an attentive examination of the sputa, and at the necropsy in the hepatized parts. It is a veritable fibrinous bronchiolitis. But this fibrinous capillary bronchitis is differentiated absolutely from the capillary bronchitis of broncho-pneumonia by this essential characteristic: in primary frank pneumonia the bronchial exudation is secondary to, and accompanies the alveolar inflammation; in broncho-pneumonia the bronchitis of the small bronchi is, on the contrary, the primitive necessary fact which always precedes and determines the alveolar lesions.

The existence of these fibrinous concretions in the finest bronchial ramifications of the hepatized regions was first pointed out by Lobstein and Remak.[1] But it is an interesting fact, which has a bearing on the interpretation of certain stethoscopic phenomena in pneumonia, that the fibrinous exudation may ascend to a considerable height in the bronchial tubes and obstruct the large bronchi. In 1854 Widemann, of Strasburg, in his thesis, inspired by Schutzenberger, studied cases of this kind.[2] Nonat, during the epidemic of influenza of 1837 in Paris, reported ten cases of pneumonia with production of false membranes in the bronchi.[3] The theses of Cadiot (1855) and Renon, 1872) are devoted to the same subject.[4] Facts of the same kind have been communicated by Bristowe and Wilks to the Transactions of the Pathological Society of London.[5] Since the publication of a remarkable case by Grancher in 1877, a great number of similar cases have been reported in France; we may cite those of Blachez, Leroux, Henrot and that of Brissaud[6] observed in the service of Millard, where the fibrinous exudation represented a complete mould of the entire bronchial tree of the hepatized lung, from the point of bifurcation to the most minute ramifications.

The bronchial casts are sometimes massive and full, sometimes thin and hollow; they are free or adherent to the mucous membrane of the air tubes. Their color is variable, generally of a cream color, sometimes dark gray, sometimes white as a diphtheritic false membrane. In Grancher's case the bronchi were filled to their bifurcation with a fibrinous, elastic, fibrillary mould, of yellowish color, readily detached from the bronchial walls. The surface of the mould, where it covered the large bronchi, kept the impress of the longitudinal folds of the mucosa. The latter had preserved its smooth aspect and was scarcely injected; an important item, for in fact the epithelium of the mucosa was absolutely intact under the fibrinous exudation.

[1] Lobstein, Arch. Méd. de Strasbourg, March, 1835. Remak, Arch. Gén. de Medecine, 1846.

[2] Widemann, De la bronchite fibrineuse, Th. de Strasburg, 1874.

[3] Nonat. Arch. de Méd., 3d Serie, t. II., p. 214.

[4] Cadiot. Th. de Paris, 1855 ; Renon, Th. de Paris, 1872.

[5] Trans. of Path. Soc., vol. vi., 1855.

[6] Grancher, Gaz. Méd. de Paris, December, 1877 ; Brissaud, Bull. Soc. Anat. July, 1878; Blachez, Soc. Méd. des Hôp., Nov., 1878 ; Anglade, Th. de Paris, 1880 ; Petit, Th. de Paris, 1881; Leroux, Arch. Gén. de Méd., 1881, t. I., p. 471; Brissaud et Beurrmann, Arch. Gén. de Méd. t. I., p. 164.

Lesions of other Organs.

The lesions of other organs than the lungs appertain to the infectant pneumonias. We should nevertheless except the bronchial glands, which are tumefied, softened, and injected, even in the localized pneumonias, and whose alteration is as constant in fibrinous inflammation of the lungs, as the alteration of the sub-maxillary glands in diphtheria of the throat.

The lesions provoked by the infectant pneumonias ought to be divided into two classes according as they are manifestly dependent on the localization of the pneumonic coccus, or as they have only an indirect or doubtful relation to the latter. In the first category we range the fibrinous inflammations of divers serous membranes, such as the pericardium and the meninges, where the micrococcus has been certainly observed; in the second, the bacterial lesions of the liver, of the kidneys and of the spleen, which are common to all the infectious diseases.

Specific Fibrinous Lesions.—These lesions are the result of the transportation of the parasite by the lymphatic system. This extension takes place by contiguity in the case of inflammations of the pericardium and peritoneum; as for the affection of the meninges, we must admit a more general infection and the penetration of the coccus into the great lymphatic circulation.

Bouillaud regarded pericarditis as a frequent complication of pneumonia, especially when the latter is seated on the left side. He, however, did not observe it but twice in twenty-six cases. Grisolle noted three cases out of fifty-eight. In the statistics of Vienna, we find pericarditis in one per cent. of the cases; in the statistics of Stockholm we find it in two per cent., in those of Bâle, in four per cent. On the other hand, in the epidemics of Frankfort and Moringen, Rodman and Kuhn insist on the frequency of the lesions of the pericardium. The first witnessed pericarditis in a fourth of the autopsies; the latter gives the proportion of sixty-three per cent. in the first epidemic, and in the second, out of eight autopsies he observed pericardial inflammation in five. These latter data agree with the result of inoculations made in animals where fibrinous pericarditis is found to be a frequent accompaniment; this was especially the case in the experiments of Talamon. It is probable, moreover, that apart from cases ending in death, the pericarditis will generally escape the observation of the physician; first, because he does not seek for it in pneumonia with the same persistence as in articular rheumatism, and in the second place because the friction sound is easily masked by the morbid bruits of the inflamed lung.

Meningitis is a less frequent localization; it is wrongly described as a purulent inflammation; it is a fibrinous meningitis, for the exudation differs in nothing, as far as the histological constitution is concerned, from the false membranes of pleurisy or of pericarditis; it is a fibrinous network imbedding in its meshes leucocytes and granular cells. The inflammation occupies ordinarily the upper and anterior regions of the brain, being a meningitis of the convexity. It may, however, extend to the entire encephalon, and gain the regions of the base, sometimes even it invades the meninges of the spinal cord. You have then the lesions of cerebro-spinal meningitis, but in order to explain these facts we see no reason to assume the supervention of any other disease, or to suppose the advent of cerebro-spinal typhus to complicate the pneumonia. As for the supposition that the epidemic typhus itself may be due to the pneumonic coccus localizing

itself primarily in the meninges, this is a question useless to bring up here, and which cannot be settled without the positive discovery and identification of the specific parasite in the exudation of the true epidemic cerebro-spinal meningitis.

We have not very abundant data to establish the frequency of meningitis in the course of pneumonia; out of 300 cases Grisolle has noticed it but eight times. The isolated observations of Zimmerman, of Surugue, of Salvy, of Laveran, Barth and Poulin, of Firket,[1] do not enable us to determine the proportion. Nevertheless in the epidemic of Moringen, Kuhn, in twenty-four pneumonic patients, observed eight that were complicated with exudative meningitis.

Parenchymatous Lesions common to all Infectious Diseases.—The infectant pneumonias, like the other parasitic diseases, determine in the parenchyma of various organs certain alterations, of which we shall not discuss here the pathogeny. The liver and the spleen are congested and augmented in volume, the kidneys are tumefied, and present to the microscope the lesions of acute diffuse nephritis. Are these lesions the consequence of the hyperpyrexia, of the direct irritation of the parasite, or of a special chemical product fabricated by the microphyte at the expense of the materials of the organism, and poisoning the nutritive liquids? We know not, and the data for serious discussion are lacking. The renal lesions are very likely produced, as Bouchard thinks, by elimination of the parasite. But rigorous demonstration is not yet forthcoming, for the round coccus found in the albuminous urine, or in the renal cells, does not reproduce the microphyte such as we observe it in the pneumonic exudation. As for the cardiac lesions indicated by Jurgensen, we cannot attribute as much importance to them as the German pathologist. In his nineteen autopsies of pneumonic patients, Jurgensen declares that he found fatty degeneration of the myocardium in all. These alterations ought to be considered as anterior to the pneumonia. Hamburger, in seven cases where the state of the heart was particularly studied, observed absolute integrity of the cardiac muscle. What does undoubtedly result from the pulmonary lesions, is dilatation of the right heart, and its almost constant obstruction by voluminous fibrino-cruoric or fibrinous clots. Despite the assertion of Bouillaud, the endocardium is ordinarily healthy; the redness of the valves is the result of cadaveric imbibition, common to all the infectious diseases, and not of inflammation. Nevertheless in several cases of infectant pneumonia, observers have noted an unquestionable vegetative endocarditis, localized sometimes on the aortic, sometimes on the mitral valves.

Anatomical Characters of the Broncho-pneumonias.

The lesions included under the name of broncho-pneumonia, present themselves under divers aspects, whose interpretation has been from all time the subject of lively discussions. Of these lesions, some purely mechanical, such as emphysema, atelectasis, congestion, ought to be studied apart as concomitant alterations; the others, essentially inflammatory, constitute the very basis of broncho-pneumonia; namely the bronchitis, the splenization and the nodules of hepatization.

[1] Zimmermann and Heller, Deutsch. Arch, f. Klin Med. Bd. V.; Surugue, Th. de Paris, 1875; Laveran, Gaz. hebd., 1875, p. 723; Barth and Ponlin, Gaz. hebd, 1879, p. 310; Salvy, Th. de Paris, 1881; Firket, Ann. de la Soc. Méd. de Liége, 1880 and 1883.

Splenization.—At the autopsy of children affected with measles or croup, and dying with the signs of pulmonary inflammation, you habitually find both lungs, especially in their infero-posterior regions, of a vinous or violaceous color; the tissue is more resistant in these parts, without, however, offering the hardness of hepatization; it only partially sinks in water. If you inflate the lungs, these characters disappear, and the tissue takes again its normal aspect. The vinous coloration and the augmentation of consistency may occupy a whole lobe or a large part of a lobe; in other cases the diseased parts are disseminated under the form of circumscribed roundish or elongated patches, in the midst of tissue which presents a rosy red or emphysematous appearance, and by the side of other bluish patches formed of collapsed or atelectatic lung. At times, too, if the lesions occupy the deep portions and not the superficies of the lung, it is mostly by palpation that you become cognizant of denser portions of tissue in the depth of the parenchyma. On section, you see nevertheless that the anterior portions of the lung are sound; the lesions remain limited to four or five centimetres of the posterior border. The surface of section of the diseased parts is smooth, without granulations; a reddish and rather thick liquid oozes from the incision. Finally, on pressing upon the lung, you force out of the small bronchi compact drops of muco-pus, and in case of croup, flakes of fibrinous false membrane. This is the appearance of the lesion described under the name of splenization of catarrhal pneumonia. This is the generalized lobular congestion of Legendre and Bailly, who refuse to see here the characters of an inflammation; it is the form of broncho-pneumonia for which Joffroy has proposed the name of spleno-pneumonia. According to Roger and Damaschino, it is the first stage of lobular pneumonia, the stage of *engorgement*.

The question whether splenization be really an inflammatory lesion or not, is not yet solved according to some authorities, who regard this lesion rather as a matter of congestion than as a variety of the first degree or of the second degree of pulmonary inflammation. Charcot has made an ingenious comparison between splenization and the epithelial lesions observed in glands whose excretory duct is obliterated; the obstruction of the bronchi by muco-pus having here the essential and pathogenic rôle. Joffroy, who adopts this view, none the less, from the point of view of subordination of the lesions, separates splenization from nodular hepatization. They are two different lesions; they ordinarily march side by side, but are not necessarily interdependent, and the splenized parts do not necessarily end in hepatization. Microscopically, the alterations do not differ from those of the engorgement stage of fibrinous pneumonia, except by the greater number of epithelial cells desquamated and obstructing the alveolar cavity; you still find there red globules and a few white globules. This is all that you observe on sections made hap-hazard. But the important fact, which directly links splenization, of whatever nature, to the processes of broncho-pneumonia, is the constant presence, as indicated by Charcot, in the midst of the splenized parts, of little nodules of induration developed around the bronchioles.

It is not always easy to distinguish these nodules by the naked eye, yet on looking attentively with or even without the help of a lens, in the direction of the centre of the lobule, you will distinguish, in the midst of smooth and even portions of parenchyma, little grayish points arranged sometimes in the form of minute clusters. These points, when they have acquired greater dimensions, appear like pearly granules; they are dense, fri-

able, and on being scraped exude a thick and grayish liquid; they resist insufflation, while the splenized parts under inflation regain their normal aspect.

In a section of a splenized pulmonary lobule, you behold these nodules under a low power sharply marked off here and there in the midst of alveoli stuffed with endothelial cells; the centre of each nodule is formed by the section of an arteriole and small bronchus. Under a higher power, the bronchus, of which the epithelium may remain intact, is seen filled by a coherent greenish mass. The wall is infiltrated with embryonal nuclei, which are diffused in variable quantity throughout the connective tissue of the vicinity. All around, except ordinarily on the side corresponding to the arteriole, are seen a certain number of alveoli filled with an exudation constituted by a network of fibrine filaments imbedding in its meshes leucocytes and epithelial cells. In cases where the lesion is just at its commencement, there exist only one or two rows of these alveoli, while the zone of splenization extends all around. These states of inflammation represent what Charcot calls the *peri-bronchial nodule*. It is the true characteristic lesion of broncho-pneumonia.

Hepatization.—The extension of these centres of peri-bronchial inflammation gives to the inflamed parts the aspect of the red or gray hepatization of frank pneumonia. In the midst of the splenized and smooth parts, the nodules, which are absolutely dense, incapable of inflation, and which sink in water, present a grayish or grayish-yellow tint; the surface of section is granular, as in true pneumonia. When the granules of hepatization are minute and disseminated, they may sometimes be mistaken for tubercle granula; this is the *partial gray hepatization* of Damaschino.

Under the microscope, the lesions are those of the peri-bronchial nodules, the number of alveoli stuffed with fibrinous or fibrino-purulent exudation is only much more considerable; in many of the alveoli, even, nothing is longer to be seen but leucocytes and granular matter. At the same time the embryonal infiltration is well marked around the bronchus and the central arteriole; it invades also the peri-lobular connective tissue stroma. There is a marked thickening of this stroma, which renders more distinct the lobular aspect of the lungs. This thickening is due, according to Charcot and Balzer, to several causes: 1. to vascular congestion and dilatation of the capillaries; 2. to infiltration of the connective tissue by a fibrino-albuminous exudation rich in leucocytes; 3. to the dilatation, at times considerable, of lymphatics, whose cavity is filled, sometimes by a fibrinous coagulum, sometimes by a mixture of fibrine and blood globules. It is easy to understand that when the inflamed lobules occupy the sub-pleural regions, the extension of this lymphangitis and of this interlobular inflammation is not slow to gain the serous membrane itself. Hence the production of a limited pleurisy, sometimes appreciable only to the microscope, sometimes characterized to the naked eye by the appearance of thin fibrinous false membranes.

Mechanical Accessory Lesions.

To splenization and hepatization are constantly joined, in lungs affected with broncho-pneumonia, divers alterations of an order simply mechanical —to wit, emphysema, atelectasis, congestion, sometimes even hæmorrhages.

Emphysema.—Emphysema is especially observed in the superior and anterior parts of the lungs; is also seen around inflamed lobules. It

may be simply vesicular, sometimes, however, there is rupture of the alveoli and escape of bubbles of air into the interlobular or sub-pleural tissue; hence the name interstitial emphysema. This emphysema belongs to the category of transient emphysemata, due to tumefaction of the alveoli; the latter, yielding by reason of their prolonged distention, and the augmentation of the intra-alveolar pressure during fits of coughing, or efforts of impeded respiration, dilate and sometimes burst. The distention may be pathogenetically associated with both the inspiratory and expiratory act—the exact measure of causal agency to be attributed to either not being absolutely determined.[1] This emphysema, then, derives nothing special from the broncho-pneumonia, and the same alterations, due to essentially the same causes, are seen in children dead from croup or whooping-cough without any inflammatory lesion of the lungs whatever.

Atelectasis.—We may say almost the same thing with regard to the atelectasis, despite the part which certain pathologists have attributed to it in the development of the lesions of broncho-pneumonia. Its mechanical origin is indisputable; we have here the same modification of tissue as the *fœtal state*, described by Jorg, in the new born infant that has never breathed, or as the pulmonary collapse attending pleuritic effusions; the mechanism only differs. In the "fœtal state" the air has never penetrated the alveoli; in pleuritic collapse of the lungs the air has been driven out by the compression exercised by the liquid; in the broncho-pulmonary collapse it is in the bronchus that the efficient cause is seated. As Gairdner has shown, it is by the bronchial obstruction that the atelectasis is explained, whether we adopt in its entirety his ingenious theory, or whether we admit the resorption, by the blood, of the air imprisoned in the alveoli.

Legendre and Bailly have well described this lesion; they have succeeded in differentiating this "fœtal state" from lobular pneumonia, and in establishing the fact that we have not to do here with an inflammatory lesion, but with a simple mechanical alteration. We cannot do better than quote their description: "The sunken tissue is fleshy, compact, but supple, lax in texture, of a specific gravity heavier than that of water, which causes it to sink in that liquid. You distinguish perfectly on the surface the cellular interstices which separate the lobules. The color is generally a reddish violet, but it may become quite dark when the blood with which it is engorged is more abundant. Its section is smooth and uniform. By pressure you cause more or less sanguinolent serosity to ooze. Lastly, insufflation causes the air to penetrate all the vesicles and restores to the organ its physiological characters." The seat of the atelectasis is the same as that of splenization, namely the circumference of the base of the lungs, the posterior regions; the tongue-like anterior portion which covers the heart is almost always in a state of collapse, sometimes the right median lobe is atelectatic through its whole extent.

The fœtal state is moreover differentiated from splenization by the following characters: The sinking of the collapsed parts is more pronounced. The color, instead of being a mahogany brown, is violet-blue or dark blue. Insufflation may be practised to any extent, while the splenized lobules oppose always a certain resistance; the consistency is more flesh-like, more firm. The grayish granular nodules are never observed; the pleura is never altered at the surface of the atelectatic parts. In fine

[1] See Phtisie Bacillaire, by G. Sée, p. 343 et seq.

the microscope shows the alveoli empty, collapsed upon themselves, while in splenization they contain desquamated epithelial cells and leucocytes.

Legendre and Bailly make no difference between atelectasis and splenization; they include the two lesions under the name "fœtal state" and clearly demarcate them from lobular pneumonia; the former being nothing but mechanical lesions, consequences of the bronchitis. If we adopt the comparison suggested by Charcot between splenization and the epithelial alterations which take place in glands whose excretory duct is obstructed, we find ourselves to a certain extent in sympathy with the views of Legendre and Bailly. The atelectasis being the first consequence of the bronchial obstruction, the splenization would be a more advanced stage of the lesion; there would be at first simple mechanical sinking, or collapse of the alveolar cavities, then alteration of their epithelial lining. It will be seen that this view differs from that of Legendre only in the affirmation of the inflammatory nature of the splenization. But this inflammation is of a quite different character from the true broncho-pneumonic phlegmasia which circumscribes itself in the peri-bronchial noduli.

Congestions and Hæmorrhages.—Congestion is a lesion which may be said necessarily to accompany every case of broncho-pneumonia. Damaschino has shown in atelectasis the remarkable engorgement of the capillaries, making a sort of hernia into the empty cavity of the alveolus. On the other hand, the passive congestion of hypostasis in dependent regions is to such a degree confounded with splenization that it is almost impossible, even with the help of the microscope, to differentiate the one from the other, and in typhoid fever one is often embarrassed in endeavoring to determine where the congestion ends and the hypostatic pneumonia begins. As for those active congestions, to whose mobility Cadet de Gassicourt assigns so large a part in the clinical history of broncho-pneumonia we shall not speak of them here, their anatomo-pathological history being so incomplete.

The intensity of the congestion may go to the extent of causing rupture of the capillaries and the formation of hæmorrhagic foci, which may occupy the pulmonary tissue or the sub-pleural regions. When the hæmorrhage takes place in the nodules of broncho-pneumonia, these nodules take on the aspect of an apoplectic infarctus; Bouchut and Labadie Lagrave in fact do not hesitate to consider them as such, and explain them as the result of emboli projected from the right heart. On the pleura the hæmorrhages appear as punctiform ecchymoses, covering a greater or less extent of surface, and similar to the sub-pleural ecchymoses observed in persons that have died of suffocation. Asphyxia plays evidently an important part in the production of hæmorrhagic foci; we must add also the changes in the texture of the capillaries, and the alterations effected in the blood. It is especially in the broncho-pneumonias of the rachitic and diphtheritic that these hæmorrhages are observed; Parrot has noted thirty cases in measles, and fourteen in diphtheria.

Anatomical Forms and Pathogeny of the Broncho-pneumonias.

The anatomical lesions which we have just described may be combined in different ways, so as to present at the autopsy a certain number of aspects which we may reduce to three principal divisions; these divisions were first proposed by Barrier as best corresponding to the sum of the facts. We admit, then, a generalized broncho-pneumonia, a dissemi-

nated broncho-pneumonia, and a lobar or pseudo-lobar broncho-pneumonia. In these three forms, moreover, we may recognize the three stages of engorgement or splenization, of red hepatization, and gray hepatization.

Generalized Broncho-pneumonia.—This form corresponds to that which Joffroy has described under the name of spleno-pneumonia, and to the generalized lobular congestion of Legendre and Bailly. It is the most common form of broncho-pneumonia. What predominates here, what characterizes this form, is the extent of alterations which appertain essentially to the type of splenization, and of fœtal state. The lungs are voluminous, distended by emphysema in their superior and anterior portions, sunken by atelectasis in their posterior and inferior parts, and present on section the special consistence of splenization. The very generalization of these lesions is incompatible with a long duration of the disease, and by reason of the habitual rapidity of the evolution, the essential phenomenon, the peri-bronchial inflammation remains, so to speak, scarcely more than outlined. With considerable difficulty appreciable to the naked eye, the grayish granules which characterize it, ought to be sought with care—else they will escape notice—in the midst of the splenized parts. Under microscopic examination they never fail to appear, and are easily recognizable. In certain cases, moreover, the disease is sufficiently prolonged to enable the peri-bronchial nodules to become prominently developed, and to extend. You see then, on section of the splenized regions, the hepatized parts irregularly mapped out, with their induration, their grayish coloration, and their granular surface, and bearing a striking resemblance to the hepatized lung of frank pneumonia.

Disseminated Broncho-pneumonia.—This has been also called lobular mammillated pneumonia, and broncho-pneumonia with disseminated nodules. The lobular dissemination of the lesions is here much more clearly marked. In the midst of the pulmonary parenchyma of a normal rose gray, are found indurated nodules of a volume varying from that of a hazel nut to that of a walnut, of a lively red or mahogany red color. Roger has compared these hard masses to the nodosities of erythema nodosum; they are found under the pleura on the inferior or anterior borders of the lungs, often in the deeper portions of the parenchyma, and especially around the large bronchi in the vicinity of the bifurcation. Each of these nodules undergoing evolution on its own account, comprehends in itself all the alterations of broncho-pneumonia, and presents in its turn the different stages of the inflammation. One of these nodosities will be simply splenized, studded only with several grayish granules resisting insufflation. In another the induration will be more pronounced and more extensive; the nodule more resistant, hepatized, does not let itself be inflated; in the central parts are seen granulations jutting upon the surface of section like those of fibrinous pneumonia. In a third stage the centre of the nodule, having become more friable, has taken a grayish yellow tint; by pressure you cause a purulent liquid to ooze from those parts which correspond at once to the bronchus and the peri-bronchial alevoli; this is gray hepatization.

Lobar or Pseudo-lobar Broncho-pneumonia.—The splenization or induration occupies in this form the greater part of one lobe, sometimes a whole lobe. As long as it is only a matter of splenization, the smooth aspect of the tissue, on the one hand, and the readiness of inflation on the other, suffice to differentiate the lesion from true lobar pneumonia. But when the broncho-pneumonia is in the stage of red or gray hepatiza-

tion, the distinction between it and the induration of fibrinous pneumonia is sometimes very difficult. In general, nevertheless, the surface of section is not as uniformly granular as in the latter; the grainy grayish aspect is disseminated in irregular islets and the granulations are much finer than in the indurated stage of ordinary pneumonia. Moreover it is rare not to find in the midst of the pseudo-lobar hepatization, lobules in various stages of change, which gives to the cut surface a marbled aspect, variegated with numerous different tints which are almost characteristic. In fine, when a lung presents a lobe in the state of hepatization, it is the rule that disseminated nodules of broncho-pneumonia exist in the opposite lung. In cases which are too complex there remains as a last resort the histological analysis.

Whatever, moreover, may be the macroscopic or microscopic aspect which the alterations of broncho-pneumonia present at the autopsy, their mode of development is the same in all cases. It is the bronchitis which dominates and directs them, generalizing or disseminating the lobular inflammation. The obstruction of the bronchial tubes, on the one hand, and the stagnation in these pipes of irritant liquids on the other, explain the double process which ends in the congeries of lesions grouped under the name of broncho-pneumonia. The obstruction of the bronchi has two consequences; the one immediate, temporary, mobile, *i.e.*, collapse of the lobules, fœtal state, atelectasis; the other of later date but more enduring, and of irritative nature, viz., splenization. The stagnation of irritant liquids containing morbific agents, micrococci, whether specific or not, has for its result the inflammation of the terminal bronchus and the alveoli which immediately border on it, and this inflammation spreads by contiguity to the neighboring parenchyma, and even to the surrounding connective tissue, ending in purulent or fibrino-purulent infiltration of the greater part of the lobule.

CHAPTER XVI.

CLINICAL DEPARTMENT.

Ordinary Type of Primary Localized Pneumonia.

Primary fibrinous pneumonia being always in our belief a parasitic infectious disease, according as the microphyte remains localized in the lungs or invades the neighboring organs and the rest of the economy, we shall describe two great varieties: primary localized pneumonia, and primary invading, complicated infectant pneumonia.

Prodromes and Mode of Invasion.

Pneumonia surprises the individual in a state of perfect health; such is the classic statement, and such is in fact the rule in a great number of cases. But this rule has nothing absolute about it, and in very many instances may be noted, if sought for, certain prodromic phenomena announcing the invasion of the disease. Grisolle has found prodromes in twenty-five per cent. of pneumonic patients interrogated by him with reference to this point. Attempts have been made to constitute of these prodromes the peculiar attribute of grave, so-called typhoid pneumonias; without doubt precursory symptoms are oftenest met with in such cases, but the prodromes belong also to the ordinary type of frank pneumonia.

Generally patients indicate as the commencement of their sickness the moment when the initial chill or the pain in the side obliged them to cease their occupation. The vehemence of the onset here effaces all remembrance of the antecedent less pronounced malaise. But this malaise unquestionably occurs in very many cases, and one might be warranted in supposing it to be a constant phenomenon, but often so slight a symptom as not to evoke the attention of the patient. It is well known how difficult it is ordinarily to determine the exact onset of typhoid fever, from the fact that the morbid invasion is here gradually progressive, and the disease reaches little by little its climax without any capital symptom supervening as a way mark. In pneumonia the severity of the chill absorbs the attention, but one can hardly suppose that some kind of preparatory process of local order did not precede the chill and the pain. This process may have been going on unconsciously, and the patient is then said to be smitten in full health. When it manifests itself by abnormal subjective signs, noticed by the patient, either because more attentive than ordinarily to the state of his health, or because his susceptibility to the least pathological modification is great, we ascertain that the chill was preceded by morbid sensations whose duration was more or less variable. Grisolle gives as the extreme terms, from several hours to one or two weeks; in general the precursory malaise is not prolonged beyond several days.

These prodromes are those of every acute disease, consisting in a general ill defined discomfort, a lassitude of all the members, painful sensations in the muscles, kidneys, nucha; sometimes in insomnia, cephalalgia, epistaxis and want of appetite. Occasionally vague pains are felt in one side of the chest: finally a little tracheitis or bronchitis may precede the pneumonia, without authorizing one to conclude from this symptom, as some authorities have done, that the affection is of influenzal nature.

Whether the patients complain of prodromes or not, a violent chill marks the outbreak of the general symptoms and of the fever. This chill has all the intensity of the chill of intermittent fevers; shivering with chattering of the teeth lasts ordinarily half an hour or an hour; we have seen it last two or three hours. Ordinarily single, the chill may be replaced by a series of slight shiverings. It is sometimes accompanied with vomiting. Very soon afterwards, generally in the first two hours which follow, appear the pain in the side, the cough, and the difficulty of breathing, which fix the attention on the lungs and reveal the cause of this intense febrile perturbation. In a certain number of cases, however,—in one-fifth according to Grisolle, the pain in the side is the first symptom of the pneumonia, being almost immediately followed by a general chill.

Functional Troubles and Subjective Phenomena.

The three principal symptoms of this order are furnished by the respiratory apparatus; these are pain, dyspnœa, and cough, with the characteristic expectoration.

The Pain.—The pain is but rarely wanting, except in the case of old people. It is localized under the form of *stitch in the side*, or more or less diffused and spread about, and indicates the affected side. It is not, however, always in exact relation with the point of the lung inflamed. Its ordinary seat is the mammary region, but it may occupy the base of the thorax as well, or the supra-spinous region, with irradiation to the corresponding shoulder. This last seat of pain is habitually met in pneumonias of the superior lobe, although even in these cases the pain in the side may be infra-mammary. Sometimes the pain is diffused through the whole of the anterior part of the affected side. This pain is exasperated by deep inspirations, and this explains the short and trying respirations of the pneumonic; it is also aggravated by the cough and by pressing over the intercostal spaces. Its intensity is variable, sometimes scarcely noticeable except during efforts of coughing, at other times so keen that the aspect of the patient expresses extreme suffering and anxiety. The pain in the side ordinarily appears almost at the same time as the chill; sometimes it is later; the patient complaining of it for the first time the next day. It disappears quite rapidly, at the end of several days, and readily undergoes alleviation under the influence of wet cups or of morphine. According to the majority of authorities, the pain is explicable by the concomitant pleural inflammation; Beau thinks it to be the result of an inter-costal neuritis seated in the posterior part of the nerve at its point of contact with the pleura. According to Jurgensen, it is due to the inflammatory distention of the lung and the pressure thus exercised on the pleural nerves. In reality it is a muscular phenomenon.

The Dyspnœa.—The acceleration of the respiratory movements is apparent from the onset. It may be the sole indication of the dyspnœa, which manifests itself only by the rapid breathing and the beating of the

alæ nasi; the patient experiencing no subjective respiratory embarrassment, no sensation of oppression. But this rarely happens. Ordinarily the increased frequency of respiration is accompanied by a sensation of oppression and suffocation which may amount to anguish. Contrarily to the pain, the dyspnœa is aggravated as the disease progresses, and its maximum may be reached the evening before the defervescence. If the fever and pain may be said to account in part for the acceleration of respiratory movements at the initial period, the dyspnœa of the later stage, when the disease is at its height, and which sometimes amounts to a veritable suffocation, can only be explained by the extent of the lesion, the spread of the congestion, the engorgement of the right heart; a certain share to be also attributed to the fever, and the asphyxiating modifications of the insufficiently oxygenated blood. It has been said that the intensity of the dyspnœa is not proportioned to the extent of the pneumonia, especially in pneumonias of the apex. This is possible, and is explicable by the very multiplicity of the causes of the respiratory oppression. But in a general way the relationship is undeniable.

The Cough and Expectoration.—The cough is also an early symptom, ordinarily synchronous with the pain in the side. It is generally painful, frequently repeated, short, and sometimes in paroxysms. These paroxysmal coughs are more frequent in the pneumonias of the apex, but Grisolle affirms that, according to his observations, cough occurring in fits is quite as common in the pneumonias of the base. At first dry, it soon is accompanied by the typical expectoration, which is noticed from the first or second day.

The pneumonic sputa are so characteristic as often to suffice of themselves to establish the diagnosis. It is their color and their consistency which constitute their pathognomonic value. They consist of an extremely dense viscous mucus, which the patients expectorate with much difficulty, and which adheres so firmly to the bottom of the spit-cup that one may invert the cup without displacing it. The color of the sputa varies, according to the quantity of blood which they contain, from yellow to bright red. The yellow sputa are called *barley-sugar* sputa, *apricot-jelly sugar*. The reddish sputa are the brick-dust or rusty sputa.

These are the only characteristic sputa, but the expectoration of pneumonic patients may present other aspects, without speaking of the mucopurulent, or salivary or pharyngeal sputa, which are ordinarily mingled in the spittoon with the rusty expectoration; you may also observe a greenish or brownish tint, but this is not very common. The color is sometimes due to biliverdine, in pneumonias with jaundice. It may also have for its cause the presence of certain parasites, as in the observation reported by Cornil to the Society of Biology in 1868. The microscope showed in this case, which is, by the way, unique, certain corpuscles presenting a very brilliant green color, and arrayed in quadrilateral masses; these corpuscles resembled sarcinæ by their disposition, but they were of much less diameter.

As for the brownish colored sputa, they present a special aspect which has caused them to be compared to *prune juice*. Grisolle's description gives an exact idea of them: "They are composed of a liquid serous matter of an obscure reddish, sometimes dark brown or even black color, and covered at times with a whitish froth which can be easily blown one side."

Save in the case of the prune-juice sputa, the quantity of expectorated matters is always small. Grisolle estimates at 60 grammes (2 ounces)

the weight of the yellow or rusty sputa expectorated in twenty-four hours. The characteristic expectoration may, however, be completely wanting, and this may happen, aside from infants who do not expectorate, in many cases of adults. Sometimes the patients cough up nothing but a thick colorless mucus, at other times the expectoration is purely catarrhal, and comes evidently from the large bronchi and not from the inflamed parts. Lastly, one quite often finds only one or two rusty sputa in the midst of mucous or muco-purulent expectoration. The chemical analysis of the pneumonic expectoration has been made in several cases, but the results obtained offer little interest. The most important character is its richness in chloride of sodium (Beale), the presence of sugar has been noted (Walshe). According to Bamberger the pneumonic sputa, as compared with the sputa of bronchitis, are richer in soda than in potassa; they contain no alkaline phosphates; there is a small proportion of sulphuric acid —(3 per cent. according to some, 8 per cent. according to others).

The microscopical examination reveals the presence of numerous red globules mingled with epithelial cells and large granular leucocytes. With a little perseverance one almost always succeeds in finding some of those fibrinous casts mentioned by Remak and Gubler, and which represent the mould of the finer bronchioles. It is only in these portions of the fibrinous exudation that one can be fortunate enough to discover the pneumonic micrococcus. As for the affirmations of Salviolio, of Zastein, of Ziel, etc., who pretend to distinguish the parasite even in the sputum, in the midst of organisms of all sorts from the mouth or pharynx, we regard such statements as very doubtful, and the more so that the descriptions given are not alike; some indicating an ovoid but mobile coccus, others a round micrococcus with or without capsule.

The physical characters of the sputa change as the disease progresses towards resolution or towards death. In the first event they become less and less viscous, they *roll* in the vessel, then they give place to an expectoration which is simply mucous or catarrhal. Sometimes the sputa are more abundant at the moment of resolution; but often they cease completely with defervescence, a fact which does not allow us to accept the view of Rindfleisch on the mode of disappearance of the alveolar exudation. When the case is unfavorable, or likely to terminate fatally, the expectoration becomes ropy or serous, of a dirty gray or brownish color. It is then that it takes on the prune-juice aspect, without, however, this latter modification being necessarily of fatal prognostic import. At the approach of death the suppression of all expectoration is the usual fact.

Physical Signs.

Period of Engorgement.—According to Laennec the first sign furnished by the physical examination of the chest is a stethoscopic sign—the crepitant râle. He taught, moreover, that a slight degree of dullness under percussion is obtained only when the engorgement is very extensive. According to Grisolle, however, from the very moment that the respiration is enfeebled, and for a still stronger reason when first the ear distinguishes crepitation, percussion made over the congested region indicates a lessened resonance. Wintrich, Woillez, and Jaccoud insist on the tympanitic character which the percussion sound over the engorged parts often takes on. In our work on Bacillary Phthisis we gave the correct

explanation of these modifications of sound.[1] Auscultation reveals an alteration of the vesicular murmur before the appearance of the crepitant râle. According to Stokes, Waters, and Stephenson Smith, it is a puerile respiration, an exaggeration of the respiratory movement, which by several hours precedes the crepitation. Grisolle, on the other hand, teaches that the precursor of the crepitation is a weakening of the vesicular murmur, which has also lost its purity and its softness. But the crepitant ronchus is the characteristic sign of the inflammatory engorgement of the lung. These râles break upon the ear during inspiration, or at the end of inspiration, under the form of puffs rapidly evolved, sharp crackling sounds, or numerous little fine dry bubbles. At the onset the crepiti occupy a very limited space; it is necessary to search carefully for them, at first on the level of the seat of pain, then at the inferior angle of the scapula, in the axilla, over the third and fourth intercostal spaces in front. Sometimes the crepitation is not apparent till the patient is made to cough. At other times it is not audible till later when the respiration has become bronchial, (Woillez, Grisolle).[2]

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[1] "The most intelligible rationale of the peculiar (tympanitic) tone seems to be this: If the consolidating material within or without the lung be not accumulated to such extent as to obliterate by external pressure the multitude of minute bronchi within it, these tubes (like so many miniature tracheæ) give their special resonance, conducted by the quasi solid or fluid material intervening, to the percussion note on the chest surface. If, on the contrary, the accumulation be sufficient to close up the fine tubes, the source of the tubular sound is annuled, and the resonance becomes that of the consolidating material and consolidated lung tissue combined—that is, acquires the character of the toneless type."—Walshe on Diseases of the Lungs, 3d Ed., p. 77. Guttmann thus explains the tympanicity of the percussion sound of the collapsed lung:

The tympanicity of the percussion sound of the collapsed lung, (and of the lung in all cases where the tension of the pulmonary tissue is diminished) can be accounted for, not by regarding the pulmonary cells as columns of air surrounded by membranous walls, and each one capable of entering independently into vibration,—their small size forbidding such an assumption—but rather by considering them as continuous, forming one large resonant cavity subdivided by very thin membranous septa, which are everywhere of homogeneous structure. These septa, like the relaxed walls of the intestine or stomach, can indeed reflect sound, but are themselves unable to enter into vibration, as they lack the principal condition necessary to the performance of this function,—a *sufficient degree of tension;* it is thus physically impossible to cause them to vibrate. So soon, however, as by inflation of the lung, this condition is realized, the tympanitic quality of the percussion sound is lost, because now not merely the pulmonary air, but also the tense pulmonary tissue responds to the percussion stroke, and the substance of the lung being a *solid* body, naturally gives vibrations quite different in nature from those of the *air* in the alveoli. The result is that these dissimilar sonorous waves to a certain extent neutralize each other, and render the production of a tympanitic or musical tone impossible. The percussion sound of the thorax is *tympanitic* in the following pathological conditions: 1. In cases of excavation of the lung substance; 2. In cases of accumulation of air (gas) in the pleural cavity; 3. When the tension of the lung tissue is diminished. (Guttmann, Handbook of Physical Diagnosis, Wm. Wood & Co. N. Y., 1880.) [The above is substantially the explanation given by Professor Sée.—Trans.]

[2] The modern view of the crepitant râle refers the sound to the sudden separation of the vesicular walls during inspiration, these having been closely applied to each other during expiration. The residual air has been expelled by swelling and exudation. This has been proved by experiment. See a paper by Dr. Workman in the Boston Medical and Surgical Journal, August 3d, 1876. In expiration pressure is exerted on the alveoli surrounding the exudation, and their cavities are effaced. The next inspiration restores them to their former condition with a crepitus. It is, in fact, proved that the crepitant râle has its seat in the alveoli and infundibula; it does not occur in the diseased parts, but in those immediately

Already at this period, palpation of the thorax shows a slight augmentation of the vocal vibrations on the affected side.

Period of Hepatization.—In proportion as the fibrinous infiltration extends, percussion gives a sound which is more and more dull; the dullness may be almost absolute over the hepatized regions. Above and particularly in front under the clavicle as far as the third and fourth rib the sound is often tympanitic. There is no occasion to refute the mistake of the first observer who believed in a complication of pneumothorax. The tympanism is of the same order, and is due to the same causes as the tympanism of pleurisy.

As a rule, there is exaggeration to palpation of the vocal vibrations, but this is not so constant as Monneret alleges. Grisolle, Skoda, Walshe have recognized the fact that there may be no sensible difference in respect to the vibratory *fremissement* on both sides of the chest, and that in certain cases even the vibrations may be diminished on the diseased side.

To auscultation, the respiration takes on the bronchial character. From blowing to tubular breathing, all intermediate degrees of intensity may be perceived. It is the expiratory sound which is at first blowing, then, as the hepatization progresses, the souffle, becoming metallic and tubular, extends to both inspiration and expiration. You can reproduce its *timbre* and its characteristics by blowing with some force into the fist closed into the form of a tube. When the hepatization is superficial, the blowing sound is audible without any great respiratory efforts on the part of the patient; it is heard under the very ear of the auscultator. The value of the tubular souffle is considerable; its intensity indicates the degree of hepatization, as its extent marks the limits of the lesion, and as its attenuation reveals the commencement of resolution. The auscultated voice presents the same bronchial or tubular character; it is the sniffling voice, —bronchophony. In the very area of the souffle, or in its neighborhood, you will still perceive, in the first days of the hepatization, especially during the bursts of coughing, volleys of crepitant râles, more or less thickly evolved. In the rest of the lungs the vesicular sound is normal; sometimes it is marked by a few sonorous râles due to concomitant bronchitis.

The Fever and its Consequences.

If we may admit certain periods in the evolution of pneumonia, corresponding to the development of the physical signs, the march of the fever does not allow similar divisions. The fever undergoes evolution after only one fashion from the commencement to the defervescence, which announces the end of the inflammatory process.

Temperature.—If we direct our attention only to the temperature, the thermometric curve offers us only two stages; the one which corresponds to the active period of the disease, the other which marks its termination, the defervescence. In the average ordinary type of frank pneumonia, the temperature mounts all at once during the initial chill to 40° C. (104° F.) and even higher. It maintains itself at this height, with slight morning remissions, for seven or eight days, then falls suddenly on the morning of the next day to the normal or below it. The fever is then of continued, cyclical type with critical defervescence, but there is nothing invariable about it. The continuity may be interrupted by matutinal remissions, so marked as to lead to belief in a true defervescence.

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around remaining nearly or quite healthy. Its origin is purely physical, and is due, first, to compression from without; second, to expansion from within.—Tr.

These *pseudo-crises* ordinarily occur from the third to the fifth day. At other times, on the contrary, deviations from the ordinary course manifest themselves by unexpected exacerbations and ascensions, easily explicable, however, in most cases by the discovery of a new focus of crepitant râles with extension of the tubular souffle. The degree of the fever is generally very high, varying between 40° and 41° C. (104° and 106° F.); it has been observed as high as 42° C. (108° F.). In the infant one may see a temperature of 41° C. (106° F.), for several days, without defervescence being on that account retarded or the prognosis of the disease being aggravated.

We shall not repeat what we have said with reference to the doctrine of pneumonic fever, and concerning the cyclical march of the temperature. This cyclical march seems only to represent the mean duration of the disease. The fever falls ordinarily the fifth, sixth, or seventh day; according to Jurgensen's statistics, comprising 721 observations, the fall occurred on these dates 372 times; but we must not forget that in the remaining 349 cases, the disease terminated at other epochs happening between the third and the sixteenth day.

The defervescence is the most striking character of the pneumonic curve. It is sometimes preceded the evening before by an extreme ascension of the temperature, the critical perturbation. Then in several hours the thermometer falls to 37° or 36° C. (98½° or 98° F.). It is ordinarily in the night that the crisis takes place, accompanied by profuse sweats. Sometimes there is a slight vesperal reascension after the morning fall, and it is not till the next day that defervescence is complete. In more rare cases the descent is gradual, by successive steps, called *lysis;* but defervescence by lysis is as little common in pneumonia as the critical defervescence is in typhoid fever. It does not last, however, more than three or four days.

Pulse.—The frequency of the pulse in pneumonia is in relation with the thermic elevation. It varies from 100 to 120 beats in a minute. According to Grisolle, it is in a general way proportional to the gravity of the disease. Ordinarily the pulse is at the same time strong and full. In certain cases it may become small, weak, concentrated,[1] whether because the heart is in reality debilitated as shown by enfeeblement of its pulsations, or because the engorgement of the venous system has left the arteries unfilled. Jaccoud rightly insists on the distinction of these two varieties of feeble pulse: the latter corresponds to what the ancients call the pulse of the *oppressio-virium* (oppression of the forces).

As for the form of the pulse, the principal character of the sphygmographic tracings is *dicrotism.* According to Galabin and Lépine this dicrotism is less pronounced in pneumonia than in typhoid fever. Galabin adds, moreover, as a differential character, that the pneumonic pulse supports without being *deformed* a stronger pressure than the typhoid pulse, at least in the first days; that, in the second place, the sphygmographic angles are more acute and the summit more vertical.

At the moment of defervescence the pulse becomes slow, unequal, and irregular. There is nothing about this, however, which is peculiar to pneumonia, but the fact is important to keep in mind, for it might lead one to suspect in aged individuals a myocardial complication, and in infants a meningeal manifestation. It indicates, on the contrary, a favorable termination, and the imminence of defervescence.

[1] A term applied to the pulsation of arteries when not easily felt under the finger.—Tr.

Digestive Functions.—Want of appetite is constant, and there is more or less thirst according to the intensity of the fever. Nausea and vomiting pertain to the initial period, and are, moreover, often wanting. In infants there is often diarrhœa, but in the frank pneumonia of the adult constipation is habitual. The tongue is almost always covered with a white thick fur: it may be dry, red, sticky, and even brown, like the tongue of typhus fever, especially in old people.

Nervous Troubles.—We shall limit ourselves to mentioning the headache, insomnia, and light sub-delirium which accompany every well marked febrile state. Among the vaso-motor troubles, allusion may be made to the red blush of the cheek corresponding to the affected side. Sometimes, as Lépine has remarked, this flushing with elevation of the local temperature, extends to the whole upper and even to the lower limb of the same side.

Herpes.—An eruption of herpetic vesicles sometimes accompanies the febrile movement of pneumonia, but it is a much rarer circumstance than is generally believed. This eruption has been noticed in 43 per cent. of cases by Wunderlich; Lebert gives the much smaller proportion of 13 per cent. It appears habitually about the margin of the lips, but also on the alæ nasi, cheek, ear, and even upon the genital parts, and around the anus. The herpes is by no means a critical phenomenon, as has been said so often, for if it may appear at the decline of the fever, the rule is that it manifests itself during the first few days, generally on the second or third day. It is a phenomenon common to a host of morbid states, whether of infectious origin or not, for the most trifling gastric embarrassment, a simple indigestion, and in the case of the female, the appearance of the menses, may provoke an eruption of herpes. Some have ascribed to this rash a prognostic value, and have regarded it as an indication of the benignity of the pneumonia; but pneumonias accompanied by herpes may prove fatal, having actually a mortality of 9 per cent. Some have regarded the herpes as an *outward projection* (so to speak,) of the pulmonary lesion, and find in this eruption an indication that pneumonia is only a herpes of the lung; we avow, despite the arguments of Fernet, that we cannot consent to attach any such pathogenetic importance to so slight and common-place an epiphenomenon.

State of the Blood and Urine.

The Blood.—Our knowledge respecting the modifications undergone by the blood in pneumonia has not much advanced since the time of Andral and Gavarret. What stands out the most clearly from the analyses made by these two authorities, is the augmentation in the proportion of fibrin. Out of 84 bleedings practised in the course of perfectly characteristic pneumonias, there were only 7 in which the figure of fibrine oscillated between 4 and 5. In all the other cases it exceeded the latter figure, keeping between 5 and 6 eleven times; between 7 and 10 nineteen times; between 7 and 8 fifteen times; between 8 and 9 seventeen times; between 9 and 10 and above, nine times. Andral and Gavarret have also noted the proportion of the solid materials of the serum in seven cases, but the results are not concordant; sometimes, in fact, they observed an augmentation, sometimes a diminution, though not very marked. The researches of Quinquaud show a slight lessening in the proportion of solid

materials; this diminution was more pronounced in the typhoid forms of pneumonia.[1]

As for the red globules, their number diminishes in a marked manner. According to Andral and Gavarret, the weight of the dried corpuscles falls one-fourth in the space of from three to five days. Enumeration made by means of the hæmatimeter gives a proportion inferior to the normal; this does not, however, reach the figures observed in articular rheumatism. The number of white globules is higher than in the normal state from the commencement of the stage of red hepatization. As in all the febrile affections, the "blood plates" pointed out by Bizzozero and Hayem are augmented both in number and extent. Hayem asserts that a veritable crisis of hæmatoblasts occurs toward the end of the disease. The number of these elements, always greater during the course of the affection, increases rapidly at this period, while that of the red globules remains apparently the same. This temporary accumulation of hæmatoblasts in the blood almost always attains its acme the day when febrile defervescence is complete.

Finally, according to Quinquaud, the proportion of hæmoglobin gradually falls under the influence of the pulmonary lesion: it descends to 80 and 75 per 1000; it does not commence to increase till toward the fourteenth or fifteenth day of the disease. In the grave so-called "typhoid" pneumonias, the diminution of hæmoglobin may be still more marked, the figure does not begin to rise again till at a later period, about the twenty-fifth day.

Urine.—The pneumonic urine offers in a very high degree the characteristics of febrile urine. The quantity is below the normal; it is highly acid, and of a deep wine-red color. The density is augmented. The organic matters, urea, extractive matters, and uric acid, are in large amount. There is, moreover, a diminution in the inorganic matters and especially in chloride of sodium.

a. Quantity.—The proportion of water is always diminished in the active period of the disease. It is ordinarily scarcely a pint, and may fall below this the first day or two. As the disease subsides, and amelioration is pronounced, the quantity of urine augments. After the defervescence there is a veritable polyuria; the patients void 2, 3, and 4 quarts of urine a day, and this polyuria may persist even for several weeks, but it does not generally last longer than ten days; it may, moreover, be less marked, and the quantity voided per diem may not exceed two quarts.

b. Density and Color.—The density is considerable, in the first few days it attains 1028, 1030. It decreases with the augmentation of water, and falls to 1010, 1006, and even below this during convalescence. The color follows the same march. Very high colored at first, marking 4 and 5 on the scale of Vogel, the urine becomes at a later date of a straw-yellow tint and is almost colorless after defervescence.

c. Brick-dust Deposits.—The red deposits, to which the ancients attached so much importance that they regarded them as characteristic of the urine at the time of crisis, are not in any sense peculiar to the critical period. Without doubt they are frequently observed at this time; but they are also common during the febrile stage.

d. Urea.—Urea is augmented in the course of pneumonia. The fact is incontestible. But at what moment does this augmentation attain its

[1] Quinquaud, Chimie Pathologique, Recherches d'hémalotogie clinique. Paris, 1880.

maximum? This is the point of controversy. According to the majority of authorities, this increase is especially noticeable during the first two or three days; the amount of urea may attain to five times that of the normal state, and even more, according to Parkes. Lépine, in an article in the Dictionary of Practical Medicine and Surgery, declares that these figures seem to him surprising, far exceeding, as they do, his own personal observations. It is generally admitted that the quantity of urea is proportional to the elevation of the temperature. This assertion has been shown to be false by Prof. Hertz, who bases himself on the analyses of Hepp of Strasbourg. In a general way the latter has proved that in numerous cases the febrile urine contains less urea than normal urine. The researches of Happfner are confirmatory. "The quantity of urea," he says, "is far from giving the measure of the temperature in the same individual, or for a stronger reason, in different individuals."[1] Lecorché and Talamon have given, as a result of their analyses, a still more radical decision.[2] According to these writers, and in opposition to what has been generally taught, there is diminution in the proportion of urea during the stage of augment in pneumonia. During the first three days of an attack of pneumonia, observed from the sixth hour after the chill, they noted the figures 3, 17, and 10 grammes, as representing the excretion of urea. It was not till after the sixth day that the urea reached notable proportions, attaining the figures of 41, 36, 43 grammes during the twenty-four hours.

Hence the percentage of urea, as well as the quantity of urine, falls in the first stage of pneumonia. Under the influence of the general perturbation inflicted by the disease on the functions and on the exchanges of the tissues, the oxidation of azotized matters is performed but incompletely; instead of undergoing transformation into urea, these substances remain in a state of inferior oxidation, constituting the extractive matters which are then found in excess in the tissues and in the urine.

The percentage of urea does not begin to increase till the second stage of the disease. It mounts then very high—40 and 50 grammes in the twenty-four hours. This increased elimination continues for several days after the defervescence, although in a less degree. It is in relation with the progressive re-establishment of the normal functions of the organism.

e. Extractive Matters.—If the urea does not measure the febrile heat, does this mean that the fever does not exaggerate the combustions of the economy? Evidently not; by the side of urea there exist a great number of ill-defined substances confounded under the name of extractive matters. These are the true ashes of febrile combustion; from 10 grammes, the normal figure, they mount up, under the influence of the fever, to 40 and 46 grammes. (Hepp and Hirz.) Unfortunately in pneumonia researches are lacking on this interesting point. Happfner alone has made several analyses, far too few to admit a positive conclusion. Nevertheless in most cases he has observed a notable augmentation; 18 and 20 grammes in twenty-four hours, but in some cases the normal proportion did not seem to be modified. What should be noted is that, according to the researches of Happfner, as well as according to Hepp, the curves of the urea and extractive matters follow divergent directions.

f. Uric Acid.—Uric acid is in excess in pneumonic urine; the daily proportion may exceed two and even three grammes. But as in the case

[1] Happfner, De l'urine dans quelques maladies fébriles. Th. Paris, 1872.
[2] Lecorché et Talamon, Études médicales, 1881, p. 417.

of urea, there is disagreement as to the moment when the elimination attains its maximum. Bartels, in two cases of pneumonia, has seen the relation between the amounts of urea and of uric acid, normal during the fever, augment in favor of uric acid at the moment of the crisis.

Bottio Scheube concludes from his special researches on this point, that in pneumonia: 1st, the urea and uric acid increase alike; 2d, the maximum of the two products is observed the day after the crisis.[1] Charvot, who has noted an augmentation of urea at the moment of the crisis, does not venture an opinion as to the uric acid: "As for the uric acid," he says, "the variations which it undergoes are such that we can form no conclusion concerning them."[2] Lecorché and Talamon are inclined to admit that uric acid goes on continually decreasing from the acme to convalescence, which would assimilate the curve to that of the extractive matters, and make it in consequence inversely proportional to the curve of the urea.

g. Chloride of Sodium.—When you pour several drops of a solution of nitrate of silver into pneumonic urine, the precipitate of silver chloride is scarcely apparent, and may be completely wanting. This disappearance of chloride of sodium from the urine of persons affected with pneumonia, noted for the first time by Redtenbacher, has been confirmed by Beale, by Todd,[3] and since then by all observers. According to Redtenbacher, whose researches embody eighty cases, during the whole period of hepatization, and till complete resolution, chloride of sodium is entirely or almost entirely wanting in the urine. The significance of this fact has been singularly interpreted by Beale. According to this authority, and as a result of his researches, the absence of chloride of sodium during the period of hepatization, arises from the circumstance that this salt is diverted toward the inflamed lung; when resolution takes place, and the force of the attraction ceases, all the chloride of sodium retained in the lung is reabsorbed, and reappears in the urine as ordinarily. But this absence of chloride of sodium from pneumonic urine is no more a special feature of pneumonia than divers other characters, such as the herpes, the critical defervescence, etc., which have been considered as peculiar to this disease. The same phenomenon, no less pronounced, has been found in other febrile diseases, even in those where it is impossible to admit an inflammatory exudation (Vogel, Happfner), as for instance, in the pyrexial period of paludal fever. It must, then, be admitted, simply, that the diminution of chloride of sodium in the urine in pneumonia, as in other febrile diseases, is due, partly to lessening of alimentation (which plays the principal part), and partly to elimination of the salt by other channels, such as the sweat, the dejections, and even the exudation.

h. Potassa and Soda.—The quantitative analysis of these bases in the urine has been made by Salkowski in four cases of pneumonia; no other authority to our knowledge has studied pneumonic urine with reference to the increase or diminution of these alkalies. Soda diminishes during the febrile period, while the amount of potassa is notably augmented. There is, on the other hand, a rapid rise in the percentage of soda after defervescence, while the quantity of potassa falls.

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[1] Bottio Scheube, L'excrétion de l'acide urique dans la pneumonie croupale. Arch. der Heilkunde, 1876, p. 185.

[2] Charvot, Des urines dans le cours et la convalescence des maladies aiguës. Th. Paris, 1871.

[3] Beale, Med. Clin. Trans., t. xxx. ; Todd, Clin. Lectures, London, 2d Ed., p. 285.

i. Albuminuria.—The presence of albumen is frequent, not to say constant in pneumonic urine. Rayer and Becquerel, who employed as reagents only nitric acid and heat, have remarked the frequency of coagulable precipitates in pneumonia. To-day, with the aid of more certain and more delicate methods, such as Gubler's goblet test, the aceto-picric test of Esbach, or Tanret's reagent (consisting of a solution of chloride of potassium and of mercury, acidified by acetic acid), there is not a case of pneumonia in which the clinical examiner, daily testing for albumen, will not find traces of it in the urine. The time of its appearance has moreover nothing constant about it; sometimes it is at the commencement, sometimes at the end, sometimes during the course of the disease that the albumen appears. The albuminous precipitate may, moreover, be seen one day, be absent the next, to be again manifest on the next day. It is, then, an albuminuria which is not only transient but variable. This albuminuria of the localized pneumonias must be absolutely differentiated from albuminurias connected with the diffuse nephritis of infectant pneumonia. Not only its irregular appearance, its intermittence, ought to serve as a distinction, but one does not, moreover, ordinarily observe in cases of the first kind red blood globules under the microscope, granular tube casts, or micrococci in the urine. Bouchard has even attempted to separate the two albuminurias by their physical characters, in proposing his ingenious hypothesis of retractile and non-retractile albumens; the non-retractile albumen being a dyscrasic phenomenon, and due to a trouble in the general nutrition, the retractile albumen, on the contrary, being the consequence of a renal lesion.

Although the differentiation may not always be so clear, or have so absolute a value as Bouchard first accorded to it,[1] and although it seems probable that albuminuria is always linked to an epithelial alteration of the glomeruli, the primary idea which led Bouchard to the study of this distinctive sign should be kept in view. The albuminurias which supervene in the course of pneumonia do not always recognize the same pathological cause. Some are due to the hyperthermia, or to the nervo-vascular perturbation inflicted by the disease on the function of the kidneys; ordinarily this is so. The albumen escapes in the urine because the conditions of blood pressure and quickness of the circulation are modified in and around the glomeruli, or because the epithelium of the Malpighian bodies is altered in its texture; probably both reasons are influential. We have here the most frequent cause of pneumonic albuminuria, the only one that is applicable to the interpretation of this phenomenon in frank localized pneumonia. The albuminuria of infectant pneumonia is probably due, as Kannenburg and Bouchard believe, to the action of microbes eliminated by the kidneys. What is certain is that the urine takes on in such cases the characters of the urine of acute nephritis, and at the autopsy the lesions of a diffuse inflammation of the renal substance are found. But it will not do to forget that in infectant pneumonia the kidneys may be sound, and that even here the albuminuria may be due to the ordinary causes of febrile albuminuria.

The constancy of albumen in the urine of pneumonia removes, then, all prognostic value from this sign. Albuminuria does not of itself indicate a grave pneumonic affection. It is on other characters than this that we must depend for such a prognosis.

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[1] See Capitan, Des Albuminuries transitoires. Thèse de Paris, 1883.

Critical Urine.—Such are the principal modifications presented by the urine in the course of pneumonia. Do there exist what may be properly called critical discharges of urine, in the sense in which the term was understood by the ancients? Nothing justifies this affirmation. The urine in pneumonia progressively undergoes modification from the period of acme of the disease to the termination, but it does not offer special characteristics at the precise moment of the crisis, that is to say the day when the temperature falls to the normal. If we were to admit a urinary crisis we could not make it coincide with the febrile crisis, for the most apparent phenomenon which differentiates the urine of the period of decline from that of the period of augment—the increase in the percentage of urea—is manifest several days before the thermic defervescence; the fact is none the less certain that during the days which immediately precede and which follow the fall of the temperature, the elimination of urea attains its maximum. How is this explained? According to Naunyn and Unruh, there is retention in the organism during the fever of an excess of waste materials whose elimination at the moment of defervescence constitutes the crisis.[1] Bottio Scheube affirms that by reason of the fever diminishing the urinary secretion, a part of the urea daily formed during the febrile period is retained in the economy and is eliminated only at the moment of crisis. Fränkel adopts the same view, and remarks that the excretion of urea is more abundant when there is albuminuria during the acme.[2] Lecorché and Talamon do not believe in the retention of urea in a state of complete formation. They think that the products inferior to urea, formed in excess during the fever, are not wholly eliminated; that the tissues remain impregnated with waste materials, and that it is this excess of effete unoxidized ingredients which, at the moment when the functions of the organism resume their regularity, attains to complete oxidation and furnishes the high characteristic percentage of urea.

Diazotic Urine.—There is a final character which differentiates pneumonic urine, viz., the diazotic reaction. (See page 24.) This reaction is wanting in pneumonia.

Modes of Termination.

The habitual termination of pneumonia is resolution, with more or less rapid resorption of the exudation. Death may supervene either at the period of red or gray hepatization. We may class among the exceptional facts, the termination by abscess, by gangrene, or by passage to the chronic state.

Resolution with Rapid Resorption of the Exudation.—As soon as the thermal defervescence is accomplished, whatever may be the physical signs noted, the disease is terminated. The aspect and complexion of the patient become changed at once, the respiration is less embarrassed, the cough is softer and less frequent, and the expectoration easier. Strength returns and the appetite is restored. The passage from the morbid state to the state of health is as rapid as had been the passage from the state of health to the state of disease; the rapidity of the recovery, like the suddenness of the onset, are characteristics which among the anamnestic

[1] Naunyn, Importance of the retention of urea in fever. Berlin Klin. Woch., 1884, p. 524; Uhruh, Retention of the materials of incomplete combustion in fever. Arch. f. path., Anat., 1869.

[2] Fränkel, On the critical excretion of urea. Charite Annalen, 1875, Bd. II.

signs of the disease, suffice to differentiate pneumonia from every other pulmonary affection.

The physical signs, as we have seen, do not enable us to recognize the exact moment when the inflammatory process is arrested. The tympanitic character of the dullness just prior to the defervescence, as indicated by Thomas, does not appear to us a sufficient index. The fall of the fever ought to be regarded as the earliest sign in the immense majority of cases. Auscultation reveals to us only the modifications which favor the resorption of the exudation. These modifications consist essentially in the granulo-fatty transformation and liquefaction of the fibrinous and cellular infiltration. The exudation is not expectorated, as Rindfleisch asserts, for in the majority of cases the expectoration diminishes or is suppressed after defervescence. The mass which infiltrates the alveoli can only be removed by resorption by the lymphatics, and this work of resorption demands some sort of previous emulsification of the exudation.

When the hepatization begins to soften, to resolve, the crepitation of the onset reappears. It is the *crepitans redux*, the returning crepitant râle of Laennec. The puffs are fewer in number and coarser, as a rule, than in the period of engorgement; the crepiti are moister and more suggestive of bubbling; but the essential characteristic persists; this râle is only present in inspiration. Very often it is accompanied with sub-crepitant râles of medium-sized bubbles, which are heard in both periods of respiration. At the same time, the dry, rude, sonorous, tubular breathing, loses its force and its intensity, it returns to the state of bronchial souffle, of simple blowing respiration. It is no longer heard except in expiration. Soon it disappears completely; the vesicular murmur resumes its normal characters, although remaining weak for some time.

Resolution with Slow Resorption.—It is quite difficult to fix a precise duration to the work of resorption. It is easy to understand that its rapidity depends not only on the extent and density of the hepatization, but also on the general state of the patient, on his age, on the energy of the molecular exchanges and on the activity of the circulation. In ordinary conditions in a vigorous adult, the resorption appears to demand about a week, but tedious prolonged resolution is much more frequent than is generally believed. Grisolle seems even to admit that it is the rule. "Numerous observations," he says, "have convinced me, that the greater part of patients who leave the hospital in a state of health, such that they can immediately resume their arduous labors, present nevertheless to auscultation signs indicating that the lung has not regained wholly its permeability." And in fact, out of one hundred and three patients he found but thirty-seven who, on their departure from the hospital, could be regarded as having completely recovered the integrity of their pulmonary tissue. In all the others there existed either a considerable enfeeblement of the vesicular murmur, or a souffle more or less rude in tone, or else a certain amount of crepitation or sub-crepitant râles with disseminated bubbles, or, even the râles of bronchitis. Grisolle's cases include the period between the twentieth and fiftieth days of the disease. Andral has many times observed crepitant râles, occupying a greater or less extent of lung, six weeks at least after the disappearance of all the acute symptoms of pneumonia. Others have noted the bronchial souffle persisting for two months after recovery. The resolution, however, ended in being complete, so that all idea of the pneumonia becoming chronic was excluded.

Death.—When the pneumonia terminates in death, the fever persists

without tendency to defervescence; sometimes, however, an attempt at crisis is observed, but the thermometer immediately rises again. The pulse becomes small, unequal, of an extreme frequency; the respiration is accelerated, the expectoration is suppressed, or else becomes more abundant, taking on the prune-juice aspect; it is fluid, ropy, dark, and covered in the spittoon with a frothy spume. The visage is cyanosed, with violet spots, the tongue becomes dry and black; the *facies Hippocratica* is apparent; the respiration is tracheal; the skin is covered with a viscous sweat, and the patient succumbs, sometimes in collapse, with a temperature below the normal, sometimes with a hyperpyretic temperature, the thermometer marking 41.5° to 42° C. (106° to 107.5° F.); Wunderlich has even witnessed a temperature of 43° C. (109.2° F.)

The physical signs do not reveal any modifications of the exudation; it is always the signs of solid induration of the lung, the dullness and tubular breathing.[1] The muco-crepitant râle of Stokes, the large-sized bubbling ronchus, no more than the prune-juice expectoration, indicate suppuration in the lung. Both phenomena are correlated, and depend on augmentation of the bronchial secretions and their accumulation in the paralyzed bronchi. The metallic timbre of these mucous râles is explained by the pulmonary induration in the focus of which they are produced.

At the autopsy sometimes red hepatization is found, sometimes gray, sometimes the two combined, without there being any possibility during life of telling by the physical signs which predominates. The cause of death is not the degree of the hepatization, it is above all the extent of the pulmonary lesions on the one hand, and failure of the heart muscle on the other. Except, it may be, in the complicated and infectant pneumonias, it is by asphyxia that the patients succumb.

Exceptional Terminations.—We include under this title the termination by abcess, by gangrene, and by chronic induration.

Termination by Abcess.—This termination is very rare. Out of several hundred autopsies of pneumonic patients, Laennec says that he has never more than five or six times met with collections of pus in an inflamed lung; these were inconsiderable, not numerous, and scattered here and there. We cannot understand how it is that, after having made this declaration, Laennec can assert a few pages further on, that he has in the course of a single year met with more than twenty partial pneumonias which terminated in abscess of the lung; and this is the more difficult of belief, since, as these pneumonias got well, the diagnosis could not have been made except by auscultation, and it is noteworthy that Laennec reproaches those physicians who regard these abscesses as frequent, for founding their opinion only on symptoms observed during life. It is not only true that these abscesses cannot reveal themselves except by the discharge of pus and

[1] The tubular form of respiration occurs in perfection in but one condition of lung, that of hepatization. In this form the phenomena appear to occur in a space limited to the immediate neighborhood of the part examined, and that space to be of tubular shape, cylindrical or flattened. The metallic character is highly developed, to such a degree that the sounds may, without exaggeration, be compared with those produced by blowing sharply through a brass tube; their dryness, rapidity of production and intensity are still greater than in the diffuse variety. It is in the tubular variety, too, that the sensation of air being drawn from and puffed back towards the ear is most distinctly marked. In the *diffused* form (often alone heard in pneumonia) the whiffling sounds appear to be produced with but moderate intensity, and sometimes at a distance from the ear, over a tolerably extended space. Vide Walshe, loc. cit., p. 98.—Tr.

the formation of vomicæ, but it is almost impossible to distinguish these pulmonary vomicæ from those of pleural origin. One can, then, regard as certainly demonstrated only the cases where the autopsy shows a collection of pus in a focus of gray hepatization. The instances of purulent vomicæ consecutive to pneumonia, given as examples of termination by abscess of the lung, ought to be regarded generally as cases of encysted purulent pleurisy opening into the bronchi, such as sometimes follow pneumonia.

Termination by Gangrene.—This mode of termination is still more exceptional if even it exists at all. Out of more than twelve hundred cases of pneumonia observed by Grisolle, not one terminated by gangrene. On the other hand, out of fifty cases of pulmonary gangrene, the same pathologist saw none consecutive to true pneumonia. It is probable that the facts cited as examples of this kind, that of Leyden, for instance, are nothing but cases of primarily acute gangrene of pneumonic form.[1]

Termination by Chronic Induration.—Buhl and Jurgensen deny absolutely the occurrence of chronic induration of croupous origin; the observations of Grisolle, of Andral and Charcot, nevertheless demonstrate the possibility of this mode of termination. The lung then presents the characters of red induration or of gray induration, according as the chronic state has persisted for a greater or less length of time after the acute pneumonia. The microscope shows a thickening of the alveolar walls, an interstitial pneumonia with fibroid transformation of the connective tissue; but there is never dilatation of the bronchi, as in cirrhosis of broncho-pulmonary origin. The physical signs are those of pulmonary induration; the general symptoms recall those of phthisis, but we have no need of dwelling on these facts, which appertain to the history of chronic affections of the lung.

Termination by Caseification.—This termination is inadmissible. Frank fibrinous pneumonia does not end in tuberculization. The facts given as such are mistakes of diagnosis. The inflammation, considered at the onset as fibrinous, was bacillary from the very start. The examination of the sputa does not to-day permit of any doubt in regard to this.

[1] Leyden, Berlin Klin. Woch., 1879, no. 20.

CHAPTER XVII.

TYPE OF PNEUMONIAS WITH INVADING OR INFECTANT TENDENCY.

General Description.

We distinguish these pneumonias from the localized pneumonias, placing them in a separate category, and under this head we include the pulmonary inflammations called typhoid or adynamic, and those called complicated. The pericarditis, the meningitis, the nephritis, etc., are not complications, they are divers localizations of the same cause which has determined the inflammation of the lungs. The generalization of the pneumonic micrococcus is the essential character of these pneumonias, the infection becoming more or less diffused.

As for the divers denominations applied to these grave forms of pneumonia, we reject them altogether. The word typhoid, as used to distinguish a variety of pneumonia, ought to be discarded, for not only does it indicate only one of the features of the disease (which may be wanting), but it causes confusion with pneumo-typhus (properly so called) and the pneumonias which supervene in the course of typhoid fever. The epithets *adynamic, ataxic, asthenic*, are also only applicable to certain modifications of this general state. The term *malignant* pneumonia represents a doctrinal tendency which hardly satisfies the scientific mind. The denomination *epidemic* consecrates an error, for the grave pneumonias are sporadic as well as epidemic. Lastly, the word *infectious* has become in common parlance nearly synonymous with parasitic. But every lobar fibrinous pneumonia being for us parasitic, that is to say, *infectious*, we are obliged to make use of the word *infectant* to characterize the difference which we wish to establish between the pneumonias which remain localized in the lung, and those which secondarily infect the organism. This being settled, we must add that this form of pneumonia is met with especially in epidemics, and that in these conditions it may smite its victims indifferently, whatever may be their age, sex or constitution. But it is also observed sporadically; it then attacks the debilitated, the aged, persons addicted to alcoholism, those that have been overworked; all individuals, in a word, whose organism for one reason or another offers but a feeble resistance to the invasion and multiplication of the parasite.

Clinically, infectant pneumonia reveals itself by a special state of the general forces of the economy, and by certain manifestations on the part of the various organs, liver, spleen, kidneys, meninges, pericardium, pleura; as for the local phenomena, they are the same as those of localized pneumonia.

Adynamia or Ataxia. Typhoid Aspect.—We have here the most apparent feature of these infectant pneumonias; the one that has most impressed observers. In a more or less pronounced degree, the typhoid aspect with adynamia or ataxia is almost constant. From the prodromic period the gravity of the disease may be obvious. The prodromes, imper-

fectly outlined and often unperceived in simple pneumonia, are here in most cases strikingly marked. For five or six days there exists a general malaise, feebleness of the limbs, epistaxis, sometimes even a little diarrhœa. Then rapidly, as the local pulmonary symptoms reveal themselves, this general state is aggravated. Sometimes from the first day or two, oftener towards the sixth or seventh, the aspect of the pneumonic patient recalls perfectly that of a typhoid patient in the second week. Sometimes the ataxic symptoms predominate; the lips tremble, the face is grimaced; there is subsultus tendinum, with convulsive twitchings of the limbs, delirium and restlessness. Sometimes, and this is most frequently the case, the prominent characteristic is adynamia with absolute prostration, hebetude of the expression, immobility of the features. This advanced degree of typhization is but rarely attained, and between the extreme forms and the simple debilitation which accompanies every acute inflammation, all intermediate stages may be met. A certain stupor, dryness of the tongue and lips, slowness of responses, prostration of the forces, are the most ordinary phenomena. It is only in certain epidemics that these symptoms are seen from the onset; in general they appear in the second stage of the disease. The fever is always intense, above 40° C. (104° F.); the march is continuous; but when the disease terminates in recovery, defervescence takes place as in simple pneumonia, generally, however, more tardily; defervescence by lysis is also more common than in the ordinary form.

Local Signs.—The local phenomena, as we have said, do not differ from those of simple pneumonia. The pain in the side, the cough, the dypsnœa, the special expectoration, the crepitant râle, the tubular breathing, all point to the pulmonary lesion. These signs, however, may be more or less modified according to the degree of adynamia of the subject. In cases where the typhoid state is very pronounced, the pain in the side is sometimes scarcely complained of by the patient, the cough is infrequent and feeble, the expectoration may be completely wanting. The stupor and prostration of the patient explain these variations in the symptomatology. For the same reason the physical signs are sometimes with difficulty appreciable. In practising auscultation, one may fail to hear any morbid sound over the hepatized parts; it is only on making the patient cough that a little crepitation is perceived, or some degree of harsh blowing respiration. The general prostration, the feebleness of the respiratory muscles, explain why the incomplete inspiration fails to produce a sound perceptible to the ear. On the other hand, this very respiratory feebleness and the infrequency of the cough, cause stagnation of the bronchial mucosities, whence result the mucous râles with large sized bubbles, which contribute still more to mask the signs of induration already so obscure.

Tendency to Infection.—We have here the veritable characteristic of the group of the pneumonias which we oppose to simple pneumonia; the local signs, in fact, do not present any marked difference, and the typhoid state may be wanting, or reveal itself only by a few adynamic symptoms. The tendency to infection manifests itself by the frequency of the pleurisy, the pericarditis, the meningitis, or the icterus; and by two phenomena on which we shall have now to dwell, namely: the tumefaction of the spleen and the abundance of the albuminuria.

Tumefaction of the Spleen.—Friedreich was one of the first to signalize the splenic engorgement in certain pneumonias, and he made it the proof of the infectious nature of the pneumonia.[1] But we cannot admit

[1] Friedreich: Die acute Milz-Tumor. Volkmann's Sammlung, 1874, no. 75.

with the German writer that this swelling is apparent from the onset of the disease.

The tumefaction is more or less early according to the rapidity and gravity of the infection; but it is not at all appreciable before the fourth or fifth day. It never attains large proportions; as in typhoid fever, it presents notable variations according to the subjects, but the autopsy always shows, even when percussion is not very demonstrative, a deep red, friable, tumefied spleen.

Nephritis and Albuminuria.—We have said that a form of albuminuria which may be called febrile, and which is common to all the acute diseases, is constant in simple pneumonia. It is also observed, and for a stronger reason, in the infectant pneumonias; this variety of albuminuria may be the only kind met with, for *nephritis* is not by any means a necessary consequence of infection, whether that infection be pneumonic, scarlatinous or diphtheritic. But the rule is that nephritis supervenes; at least the lesion appears constant, in cases terminated by death. (Kuhn.) When the albuminuria is a part of an acute diffuse nephritis, the urine takes on the ordinary characteristics of the urine of acute Bright's disease. The quantity is diminished, the urine becomes scanty; it has a dull dirty beef-broth color, sometimes a *dead-leaf* color, characteristic of the presence of blood. The albumen is no longer under the form of a scarcely appreciable cloud, necessitating, in order to be perceived, the employment of the most delicate tests. The ordinary test of heat and nitric acid is sufficient to cause the urine to coagulate. The albumen is then in notable quantity and when weighed is found in the proportion of one or two grammes to the quart. The microscope shows blood globules, large round cells and numerous epithelial and granular casts, whose abundance leaves no doubt as to the real lesion of the renal parenchyma. These casts are infiltrated with fat granules; round micrococci are also found there, forming masses more or less voluminous. In cases where the renal lesion is profound, the diminution of the urine may amount to a veritable anuria. (Monnusen.) Sometimes one or more attacks of hæmaturia, as in scarlatina, precede the appearance of the albuminuria.

While febrile albuminuria disappears with defervescence, the albuminuria due to nephritis persists for an indefinite time after recovery from the pneumonia. It may be accompanied with œdema of the limbs and face, like scarlatinous nephritis; but this occurrence, ordinary enough in the acute Bright's disease of scarlet fever, seems to be exceptional in the pneumonic infectant malady. At all events, it is not probable that the pneumonic nephritis ends directly in a true chronic Bright's disease. The albumen gradually disappears after a variable time, sometimes two or three months. This nephritis, then, has no gravity by itself, either as a consecutive lesion, or as an epiphenomenon of the causal disease. It indicates a state of general infection, but it is only an episode, which, by reason of its frequency, does not enable us to judge of the gravity of the infection. If, then, albuminuria linked to nephritis is an index of bad augury, by itself it cannot be considered as rendering the prognosis more unfavorable.

Pleuro-pericarditis and Meningitis.

Pleuro-pericarditis.—Pleurisy is constant in the infectant pneumonias. The lesion of the pleura is in the main identified with the first stage of the infection. This pleurisy is fibrinous, fibrino-hæmorrhagic,

or fibrino-purulent; it is more or less extensive. We shall return to its clinical characters farther on. We would only indicate here that it is almost inevitably accompanied with pericarditis, which is produced by the direct propagation from one serous membrane to another of the pneumonic micrococcus.

The frequency of the pericarditis nas been differently calculated. (See page 88.) Clinically its diagnosis is not easy: its symptoms are confounded and lost in the general phenomena of the infectant disease, and the physical signs are obscured or masked by those of the pulmonary inflammation. One may, however, succeed by careful auscultation in detecting the only certain sign, the pericardial *frottement*. Law has reported a certain number of cases of pericarditis complicating acute pulmonary affections. The pericarditis is recognized especially by its acoustic and tactile signs. But what Stokes has said with reference to inflammations of the pericardium associated with chronic diseases of the lungs, should be held true for the pericarditis of pneumonia—they are almost always latent.

Meningitis.—It is the same with pneumonic meningitis. You must not expect to find here the symptomatology, in all its striking features, of acute meningitis, convulsions, contractions, vomitings, headache, etc. The meningeal inflammation does not come on except in the last days of the disease, being an ultimate phenomenon. The patient is already at that time delirious, having been so for several days; the cerebral obtusion and the prostration are very marked when the meningitis declares itself, and the symptomatic reaction of the brain is almost nil. Hence one easily comprehends that these meningites often pass unperceived and are only detected at the autopsy. The situation is the same as in rheumatism and typhoid fever when it is a question of diagnosticating cerebral rheumatism, or the ataxic and nervous form of dothinenteritis from a true inflammation of the encephalic meninges.

A sudden sinking, with semi-comatose state coming on abruptly, intellectual obtusion, a staring look with inequality of the pupils, grinding of the teeth, trembling of the lips and hands, stiffness of the neck and limbs,—such are the several symptoms noticed in the cases of pneumonic meningitis, and which in one or two days end in complete coma and collapse. In several instances, moreover, paralysis of the members has been noted; hemiplegia according to Jaccoud and Lépine. But these last phenomena would tend to obscure rather than clear up the diagnosis.

The symptoms which we have indicated will acquire a greater value in cases where the pneumonia, having run its course thus far without any well marked nervous symptoms, is all at once complicated with cerebral troubles. But in instances of pneumonia with delirium tremens, or of ataxo-adynamic pneumonia, we must recognize the fact that these cerebral phenomena cannot be of any great diagnostic utility, for they will be confounded with the symptomatology of these morbid states; you will generally have grounds to suspect, but almost never to affirm, the existence of meningitis with exudation.

Other Localizations.

Vegetative Endocarditis.—We have here a rare consequence of the pneumonic infection, but it indicates an infection carried to the highest degree. In all the instances where it has been observed, there existed at the same time multiple localizations in most of the serous membranes.

In the fact communicated by Darolles[1] to the Anatomical Society, the endocarditis of the aortic valves co-existed with meningitis. In an observation of our own[2] there was seen along with vegetative endocarditis pleurisy and general peritonitis. The patient of Barlow presented simultaneously inflammation of the mitral, meningitis of the base, infarctus in the kidneys and spleen, and hæmorrhage of the retina.[3] In Barth's case there was also coincidence of vegetative endocarditis with fibrinous meningitis of the convexity.[4]

Arthritis.—Examples of joint inflammation in the course of pneumonia are also exceptional. Such cases are generally considered as due to purulent infection, consecutive to gray hepatization. (Grisolle, Böckel, Doléris.) We make here the same objection which we have already made with reference to pneumonic meningitis. We do not believe that we have to do in these cases with real purulent effusion, but with a puriform effusion, composed of fibrine which has undergone the granular trans. formation, mixed with lymphoid cells, and infiltrated with granulationst It is not pyæmia—purulent infection—which produces these arthrites, bur the pneumonic infection; and on examining and cultivating the articulas exudation, the pneumonic micrococcus will be found. Grisolle, who hawitnessed four facts of this kind, says that in only one case the articular pains were noticed from the very first; in the three other patients the symptoms of arthritis supervened at the decline of the disease. These are not wandering but fixed arthritides, with swelling and redness around the joint. In the majority of published cases there existed fatal pneumonia, but this is not an invariable rule, as Grisolle's observations prove.

Phlegmasia Alba Dolens.—Thrombosis of the femoral vein with phlebitis may be observed at the decline of pneumonia, or at the beginning of convalescence. This phlegmasia does not differ from that which in the same circumstances supervenes as a sequel of typhoid fever. Thrombosis of the portal vein with pylephlebitis has been noticed in grave pneumonia. (Ledieu.)

Parotiditis, Thyroiditis.—Parotiditis and even suppurative thyroiditis[5] have been noted as sequels of pneumonia. But these lesions were rather complications of convalescence than true determinations of the primary disease.

Pneumonic Icterus and Bilious Pneumonia.

Pneumonia with icterus and bilious pneumonia are two things absolutely distinct. This is a point which it is important right here to establish; considering the way in which certain authorities, and in particular the German authorities have confounded these two states.

Pneumonic Icterus.—Icterus is a phenomenon quite common in pneumonia. Grisolle says that he has observed it in 20 out of 277 patients in whom he has searched for it with care. He adds that there is no acute affection where one observes as often as in pneumonia the yellow jaundiced hue of the sclerotic and of the skin. The proportion varies with different statistics; while those of Vienna, whose importance cannot be

[1] Darolles, Soc. anat. de Paris, 1875, p. 376.
[2] G. Sée, Un. Méd., 1882, loc cit.
[3] Barlow, Med. Times and Gaz., Feb., 1881, p. 187.
[4] Barth, Revue de Médecine, 1882, p. 681.
[5] Leichtenstern.

overrated, indicate only a proportion of one per cent., those of Gerhardt give a little more than five per cent., and those of Chwostek, cited by Lépine, twenty per cent. out of 147 cases. We must, moreover, make the distinction between the simple forms of pneumonia and the infectant forms. There are without doubt differences from the point of view of frequency of the icterus in the two groups, as there surely are from the point of view of its pathogeny. Bouillaud has proposed, in order to explain the production of the icterus, the theory of a propagation of the inflammation by the way of the diaphragm from the lung to the hepatic tissue; but he does not base this view on any positive fact. The observations of Drasche, moreover, contradict this manner of regarding the derivation of the icterus. In nineteen cases, the pneumonia occupied the right lung in seven,—the base in five, the apex in one, and the whole parenchyma in one;—four times the pneumonia was double, and eight times the left lung alone was affected.[1]

Bence Jones thinks that the icterus is of blood origin, and that it is the result of a want of oxidation of the blood. Murchison inclines to admit a congestion of the liver, produced by the irritation of the pulmonary pneumogastric, transmitted by reflex action to the liver.

According to Jaccoud the icterus recognizes divers causes. It is due in certain cases to blood stasis in the system of the inferior vena cava; in others it is linked to duodenal catarrh with obstruction of the choledochus; lastly, in certain rare instances it is in relation with a diffuse hepatitis. In three autopsies, Misler has observed duodenal catarrh, and inflammation of the choledochus, with obstruction by a mucous plug.[2] Lépine says, moreover, that he has several times seen in icteric pneumonic patients, redness with swelling of the duodenal mucosa, and redness of the large bile ducts, but was not able to discover any mucous plug. His pupil Bonnet, in three cases of pneumonia with icterus, has sought by the aid of the microscope for the alterations in the liver, and has noted in all a well pronounced catarrh of the bile ducts with proliferation of epithelium, and slight embryonal infiltration around the ducts.[3]

In our opinion we ought to distinguish at least two classes of cases. In the one, which corresponds to simple localized pneumonia, the icterus is produced by sudden congestion of the liver, under the influence of the circulatory trouble which ensues from the rapid suppression of a part of the vascular area of the lungs. From this results an excessive formation of bile; and there is here a clear explanation of the fact that the stools preserve their normal color, or even take on a bilious hue. In the other class, which comprehends the infectant pneumonia with icterus, there is alteration of the liver, and this alteration pertains to the bile ducts: there is catarrhal angiocholitis of the fine interlobular ducts, as Bonnet has observed, with or without like inflammation of the larger ducts.

The icterus is not an early symptom in the pneumonia, it appears ordinarily from the fifth to the sixth day. It is generally little marked: it is the coloration of the conjunctivæ (icteroid tint) and the reaction of the urine which furnish the clearest signs. Nitric acid dropped into the urine gives the bottle-green color of biliverdine. It is, then, a biliary icterus, and not a hæmaphæic icterus.

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[1] Drasche, Œsterr. Zeitsch. f. prakt, Heilk, 1860, No. 23.

[2] Misler, Ueber biliöse Pneumonie. Deutsch Arch. f. Klin Med, 1872.

[3] Bonnet, On three cases of pneumonia complicated with jaundice, with alteration of the liver. Revue de Méd. et de Chir., 1878, p. 662.

When the pneumonia gets well, the ordinary duration of the icterus is about eight days. When the disease proves fatal, the jaundiced tint diminishes, or becomes even deeper. In cases of adynamic pneumonia, with delirium and typhoid aspect, the adjunction of this icteric hue gives to the patient the appearance of grave or malign icterus. This jaundice is quite rarely accompanied with vomiting and bilious diarrhœa; this is a matter of some importance and which almost of itself suffices to differentiate pneumonia with icterus from the bilious pneumonias described by Stoll. These symptoms, according to Grisolle, are wanting in six out of seven well developed cases.

Bilious Pneumonias.—If it is deemed best to retain this term, it is at least necessary to preserve the special signification assigned to it by its author. Now it suffices to recall to mind the views of Stoll on the fevers of summer and on the rôle of the bile in the production of these fevers, to comprehend that bilious pneumonia has nothing in common with pneumonia complicated with icterus. "Every summer," says Stoll, "the bile produces fevers which are all of the same nature, but which vary in different years in respect to malignancy, greater or less rapidity of the march, or the predominance of certain symptoms. The bilious humor, according as it is more or less acrid, more or less abundant, provokes on entering the blood, fevers of variable gravity; the bilious fever moreover may be conjoined with inflammatory or other fevers." Stoll describes a bilious pneumonia, as he describes a bilious rubeola, a bilious rheumatic fever, bilious hæmoptyses or hemiplegias. The characters of this bilious state are bitterness in the mouth, nausea, vomiting of acrid and yellow colored matters, with or without diarrhœa presenting the same appearance; lastly and especially, the yellowish color of the fur which coats the tongue. It is not at all a question of icterus in these cases. A certain proof, moreover, that Stoll did not in the least confound bilious pneumonia with pneumonia complicated with icterus, is that none of the patients given as examples of bilious peri-pneumonia (vide Ephemerides of the year 1777) presented the least jaundiced color; and on the other hand, when speaking of the inflammatory fevers of April, 1779, Stoll writes: "There were also inflammations of the liver alone or accompanied with pleuro-pneumonia. Sometimes the entire hypochondriac region was affected, sometimes the thoracic viscera participated. In a few of the patients the icterus was conjoined with hepatitis, and then the disease was very grave."

Practically, then, as well as theoretically, bilious pneumonia, in the mind of Stoll, differs absolutely from icteric pneumonia. We leave to one side the theoretical idea of the action of the acrid bilious matter; but we do in fact observe, although more rarely doubtless than in the time of Stoll, pneumonias complicated with the gastric state described by this writer; we call the latter, "summer biliousness," (*embarras gastrique saisonnier*). These pneumonias have no gravity; far from that, they seem on the contrary, to be ameliorated by one or two emetics. We cannot, then, give our sanction to the confusion which the German pathologists have made between the two varieties of pneumonia, and to which Jannsen has recently subscribed, in identifying the bilious pneumonia of Stoll with the infectant pneumonias with icterus.[1]

[1] Jannsen, De la pneumonie bilieuse. Arch. f. Klin. Med., Sept., 1884.

CHAPTER XVIII.

LOCAL ANATOMICAL FORMS.

We have thus far studied the manner in which pneumonia influences the organism, according as the parasite circumscribes itself in the lung or invades other organs and tissues. We have now to pass in review the clinical aspects which it presents, according to the depth, extension, and seat of the lesions produced, on the one hand, and according to the previous state of the subject on the other, and the conditions of resistance or vitality which the latter presents.

Abortive Pneumonias.

The characteristic of these pneumonias is the rapidity of their evolution and their short duration. They terminate in three or four days, sometimes convalescence takes place from the second day. Defervescence always appears suddenly, and the physical signs disappear with equal suddenness. These pneumonias have been designated under the names of pneumonic febricula (Bernheim), peri-pneumonic synocha (Marrotte), ephemeral pneumonia (Leube), hæmo-pneumonia (Woillez), and congestive pneumonia.

It has been said that in these cases the lesion does not overpass the first stage and that it is chiefly characterized by pulmonary hyperæmia. This affirmation can only be based on the stethoscopic signs, as recovery is constant. Now the crepitant râle, the bronchial souffle, and bronchophony are observed here as in ordinary pneumonia, although they so much more speedily disappear. There seems to be no good reason to doubt that the anatomical process is the same, that is to say, constituted by a fibrinous coagulable exudation. What anatomically differentiates abortive pneumonia from ordinary pneumonias is the not easily explicable limitation of the morbid process, both in profundity and in extent, and the rapidity of absorption of the exudation. This form of pneumonia may be observed in adults and even in the aged. (Charcot.) But it belongs especially to adolescence and the period of second dentition.

The disease begins suddenly by a violent chill followed by pain in the side, with cough and oppression. The thermometer mounts up to 104° F. and above. The expectoration may be wanting, may be simply viscous and yellow, or offer the typical brick-dust, rusty aspect of pneumonic sputa You perceive in a very limited tract several puffs of crepitant râles soon followed by whistling and bronchial respiration with bronchophonic resonance of the voice. Then suddenly, the next day, or in the course of a couple of days, at the moment when you expect to see the pneumonia spread, and the blowing breathing more marked, the disease is arrested or aborted. The thermometer falls to the normal figure, and in twenty-four or forty-eight hours every morbid local sign has disappeared.

To give to this form of pneumonia the name of acute congestion of the

lungs or of "fluxion of the chest," is to misconceive its real nature; it is a specific pneumonia, and not a congestion, just as abortive typhus, which undergoes its evolution in eight or nine days, is a typhoid fever, and not an enteritis, or an acute gastric disorder.

Relapsing Pneumonias and Recurrent Pneumonias.

It is necessary here to keep in mind the difference between *relapse* and *recurrence*. The distinction is the same as in typhoid fever. Pneumonia is said to be relapsing when it is reproduced during convalescence at a period when the latter is not yet completed. It is recurrent when the disease reappears after an interval of perfect health, a period for instance of several months or several years.

Relapsing Pneumonias.—Pneumonia with relapse is rare. The ratio observed by Briquet is exceptional: sixteen relapses in eighty-two cases.[1] Grisolle says that he has noted relapse in only one twenty-eighth of his patients.

The relapse ordinarily occurs about the fifteenth or sixteenth day of the disease. Relapsing pneumonia belongs to the type of abortive pneumonias. The fever, the rusty sputa, the crepitant râles, and the souffle reappear with the same characters as in the first stage of the disease, but defervescence takes place in two or three days: it may, however, be delayed till the fifth or sixth day. Grisolle has observed in one patient as many as three successive relapses, each one of a duration of eight or nine days. According to Briquet, relapses are more frequent in pneumonias of the apex. The short duration and the benignity of the relapses in pneumonia assimilate them, from a symptomatic point of view, to the relapses of typhoid fever.

Recurrent Pneumonias.—Like erysipelas of the face, pneumonia seems to have a great tendency to reproduce itself in the same individual. Out of 165 pneumonic patients, Grisolle found 54 who had experienced previously, at a time more or less distant, one or more attacks of the same disease. The number of recurrences in the same subject is sometimes considerable, and these repetitions seem to have a tendency to approach each other and in direct ratio to their frequency. As recurrences are multiplied, the intervals which separate them diminish. Chomel has observed pneumonia ten times in the same individual, J. Frank, eleven times; Andral cites the case of a patient who had from fifteen to twenty recurrences. Ziemssen has observed a child who had in the space of five years, five attacks of pneumonia, four on the left side, one on the right; Charcot has related the case of an old woman who from 1861 to 1867 had eight attacks of pneumonia, seven on the left side and one on the right. The first two took place in January and in May, 1861, the third and fourth in January and May, 1864, the fifth and sixth in January and April, 1865, the seventh and eighth in January and March, 1867; the patient succumbed to this last attack. The facts of Ziemssen and of Charcot show that the recurrence pertains habitually to the same lung, and the left lung seems to be the seat of predilection. The same conclusions are drawn from the statistics of Grisolle; out of 35 patients, the same lung was the seat of the pneumonia twenty-five times, and the recurrence was on the left side in the proportion of sixteen to nine.

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[1] Briquet, Arch. Gén. de Méd., 3d Series, Vol. 7, p. 481.

The symptoms of these recurrent pneumonias are those of ordinary pneumonia. A fact of importance in connection with this subject is that in the long run, at least in old people, as was proved by Charcot's post-mortem observations, these repeated inflammatory attacks determine in the localities affected the alterations of chronic inflammation, and these parts present the characters of gray or slate colored induration with sclerous thickening of the alveolar walls.

Pneumonia of the Apex.—Anatomically this variety does not differ from pneumonia localized elsewhere, for we cannot admit the opinion of Briquet, who thinks that pneumonias of the apex are more particularly connected with the existence of pulmonary tubercles. The pneumono-coccus may localize itself, without any previous bacillary infection, in the upper lobes. May it not, however, be associated with the tubercle bacillus? In other terms, may not fibrinous pneumonia co-exist with tuberculous pneumonia? This is an hypothesis which is very probable, as the different pathogenic microbes do not seem to have a tendency to exclude each other. Do we not see the septic vibrio in union with the pyogenic microbe, producing pyo-septicæmia or purulent surgical infection? The bacillus of anthrax and the micrococcus of pus may cohabit in the same subject and determine a purulent charbon. It is, then, possible that the pneumonic coccus and Koch's bacillus may unite in producing a fibrino-tuberculous inflammation; clinically, however, the detection of the bacillus tuberculosis in the expectoration will always decide the question in favor of pulmonary phthisis.

However this may be, from an etiological and clinical point of view, pneumonia of the apex offers certain peculiarities. Pneumonia in general occupies preferably the right lung; this is the case in almost two thirds of the cases. Grisolle gives the ratio of eleven to six. In the case of pneumonia of the apex, this preference declares itself in a remarkable manner.

On comparing the statistics of Barth, Briquet, Rilliet and Barthez, Behier, and St. Ange, we find that out of 103 cases of pneumonia of the apex, in 89 the pneumonia was seated in the right lung. The whole of the upper lobe may be invaded, the anterior part as well as the posterior, although in the view of certain authorities, infra-clavicular pneumonia is always tuberculous.

As for the frequency of localization at the apex as compared with pneumonias of the base, it is much less, being in the ratio of 1 to 2, according to Briquet and 3 to 4 according to Grisolle.

Pneumonia of the apex is more common at the extremes of life, in the infant and in the old man. It seems preferably to select debilitated adults. But these are conditions whose importance it will not do to exaggerate, for the same apical localization is met in the most robust, and in individuals of good constitution.

Intensity of the general symptoms and inconspicuousness or obscurity of the local signs, (at least in the first few days,)—such are the two most important clinical characteristics of pneumonia of the apex.

It is especially in the infant that the first characteristic is witnessed; as we shall see, it is in this form of pneumonia that convulsions and meningeal symptoms are most often observed, and lead astray the physician. In the adult also the febrile and nervous symptoms attain great intensity. The fever is very high, the delirium almost constant, more or less violent according as the patient is alcoholic or neuropathic. The dyspnœa is also extreme, with anguish, anxiety, suffocation, and is also remarkable in that

it is in no sense in relation with the extent of the lesion. This respiratory distress is also aggravated by the vehemence of the cough, which is oftener paroxysmal and painful in this than in the other forms.

With these so marked symptoms, the physical signs are in striking contrast by their inconspicuousness and by their tardy appearance. The expectoration is often wanting; it is scanty, mucous, and but slightly streaked with blood. Bouillaud explains this fact by the little effect that the movements of expiration and the shocks of the cough produce on the apical tissue. Percussion, very difficult in the upper and posterior parts of the thorax, cannot furnish any very clear data. We should, however, note the frequency of the infra-clavicular tympanism, which is much greater in these cases, according to Woillez, than in the other varieties of pneumonia.

As for auscultation, during the first two or three days, one may not observe any morbid modification of the respiratory bruit. The foci of crepitant râles appear at last, but very much circumscribed; it is necessary to search for them with care and to auscultate attentively: 1. the supra-spinous fossa, especially towards its external part; 2. the top of the axilla below the arm; 3. the entire infra-clavicular region. Sometimes the bronchophonic resonance of the voice, or the blowing *timbre* of the cough, reveal the pneumonia before the appearance of crepitant râles. A little later the ordinary signs are well marked, with the characteristic tubular souffle. In certain cases, nevertheless, the signs remain obscure and doubtful during the whole course of the pneumonia.

Defervescence, according to most authorities, is more tardy than in pneumonias of the base; the disease does not terminate before the tenth or twelfth day. Resolution is also much slower, and the physical signs persist longer after the fall of the fever.

In fine, we have seen that, according to Briquet, relapses are more frequent in pneumonia of the apex. Ziemssen also indicates the passage of the inflammation from the superior to the inferior lobe after an apyretic interval which lasts from several hours to several days. Sometimes there supervenes a crossed pneumonia; the pneumonic evolution having terminated in the upper lobe on one side, reappears after a variable interval in the inferior part of the opposite lung.

As for prognosis, it has been deemed more grave in pneumonias of the apex, (Andral, Monneret, Pitres). According to Grisolle the mortality, which is one in eight in pneumonias of the base, is one in five in apical pneumonias. We do not deny the gravity of pneumonia of the apex, but it is not the localization in the upper lobe which constitutes this gravity; it is rather the etiological conditions which have favored this localization of the microbe, it is the subject's previous bad state of general health which darken the prognosis. In a vigorous adult, and in children, pneumonia of the upper lobes is not more grave than basal pneumonia.

Diagnosis in young children is sometimes rendered difficult by the violence of the nervous symptoms, or by the meningitic appearance which the patient presents. We shall return to this point farther on. In old people it is the feebleness of the respiratory movements which serves to obscure the physical signs and prevent the precise determination of the seat of a pulmonary inflammation, the existence of which the general state of the patient sufficiently indicates. In fine, in the adult, pneumonia of the apex being diagnosticated, one may have to ask: 1. if it be a frank fibrinous pneumonia; 2. if it be a case of fibrinous pneumonia in an individual

already tuberculous; 3. if the physical signs point to acute tuberculous deposition; 4. if it be not an affair of acute primary caseous pneumonia? In such instances, the history of the case, the auscultation of the other lung, the march of the disease, finally, the microscopical examination of the sputa, will clear up the diagnosis.

Central Pneumonia.

Pneumonia is called central when it begins in the deeper portions of the pulmonary parenchyma, the part engorged or hepatized being separated from the ear by a certain thickness of healthy tissue. In these conditions, the pain in the side is habitually wanting, but the sudden onset, the initial chill, the rusty sputa, indicate the development of a pulmonary inflammation. Does this inflammation manifest itself by physical signs perceptible to auscultation? Laennec replies in the affirmative, but Andral and Grisolle give a negative answer. "Not only," says Laennec, "may one recognize a central pneumonia of moderate extent, but it is possible to determine that it is such. At the onset this crepitant râle is audible deep in a circumscribed point, and superficially is heard the sound of pulmonary expansion and contraction; sometimes this murmur is pure and sometimes almost puerile. When the pneumonia passes to the stage of hepatization, bronchial respiration is heard at some depth, while normal pulmonary respiration exists at the surface." Laennec affirms that he has detected by the deep crepitant râle, central pneumonias involving cubic dimensions not larger than an almond or a filbert. Grisolle, on the other hand, thinks it is impossible by auscultation to diagnosticate a nodule of induration situated in the centre of a lobe. He refers to two cases of extensive central pneumonia, where several millimetres of permeable lung sufficed to intercept and suppress the pathological *bruits* which were produced deep in the parenchyma. When you think of the stethoscopic difficulties attending infantile broncho-pneumonia, for instance, of the absence of every appreciable sign of induration, even in cases where numerous nodules of hepatization are found at the autopsy, it is impossible not to partake of the opinion of Grisolle and Andral, and it seems evident that Laennec, with somewhat of a partisan bias, has stated more than the facts of the case actually warrant.

However this may be, it is certain that with the rational signs of pneumonia, existing for three or four days, with the fever and rusty expectoration, it may not be possible in any point of the lung to find the physical proof of the pulmonary lesion. It is not till the end of several days that you discover in a limited space, a focus of crepitant râles and tubular respiration. In such cases one is justified in affirming that the inflammation must have commenced in the deeper portion of one of the lobes, and that the signs did not become appreciable till the moment when, in its progressive extension, the lesion reached the sub-pleural cortex of the lung.

Migrating or Ambulating Pneumonia.

Under this name are included pneumonias of prolonged duration, evolving by successive centres more or less distinct, and thus invading in their serpiginous march the greater part of one lung. The similarity of this evolution to that of erysipelas of the face led Trousseau to adopt the term of *erysipelato-phlegmonous pneumonia*. "The parenchymatous

phlegmasia," says Trousseau, "instead of limiting itself to the place where it was first developed, has a singular tendency to invade other parts; it takes on an ambulatory form, analogous to that presented by phlegmon of the cellular tissue called phlegmonous erysipelas."[1]

Taking up the idea of Trousseau, but pushing much farther the comparison, even concluding identity of nature from analogy of evolution. Friedrich and Kussmaul give to this form of pneumonia the name of erysipelatous pneumonia. As we shall see, this identity of nature is but an hypothesis, not at all demonstrated; erysipelatous pneumonia is a particular species which has nothing in common with pneumonia of migratory form.

The latter is a kind of frank fibrinous pneumonia, remarkable only by its spreading and invading tendency. It is not an affair of disseminated nodules of hepatization, as in broncho-pneumonia, but of a gradual and progressive invasion; the lesion extending from place to place under the form of centres of variable dimensions. Each focus thereupon reproduces the symptomatic tableau, with its period of engorgement and of induration, of crepitant râles and of souffle.

In the case of Trousseau, the central lesion, during the first few days, spread first to the surface on the level of the tenth rib in front; then it reached the axillary region, where it was arrested for some time, during which there was a temporary amelioration of the general condition. But soon a new centre appeared in the posterior part of the lobe, then the superior lobe was taken in its turn; ataxo-adynamic phenomena ensued and the patient succumbed.

It will not do to take for granted that the prognosis of migratory pneumonia is always grave. Hambürger[2] has published several cases observed in the service of Kussmaul which terminated by recovery after a duration which varied between ten and seventeen days.

Double Pneumonia.

The frequency of double pneumonia has been variously estimated. Grisolle indicates a proportion of only 6 per cent. According to the statistics of Vienna, this proportion reaches 14 to 15 per cent.; it is certainly as high as this or even higher.

The extension of the inflammation to the other lung is always a rather tardy phenomenon. The two lungs are never invaded simultaneously in frank pneumonia. It is ordinarily in the stationary period of the disease, or during its decline, that the localization takes place in the lung opposite to the one primarily affected. Grisolle, in order to explain the double invasion, supposes a sort of reflex action exercised by the inflamed lung on the healthy lung, "in virtue of that law of mutual and reciprocal suffering of double organs, of which we find so frequent examples in practice." According to this hypothesis, the secondary focus of inflammation ought to occupy the part of the sound lung symmetrical and homologous with that of the lung primarily affected. But it is generally not so, and the existence of *crossed* pneumonias, that is to say, pneumonias of the lower lobe on one side succeeding pneumonias of the apex on the other, suffices to contradict the reflex theory of Grisolle. Double pneumonia is in reality

[1] Trousseau, Clinique Médicale, t. 1, p. 884.

[2] Hambürger, Untersuchungen über Croupen Pneumonie, Strasbourg Diss. inaug., 1879.

but a variety of migratory pneumonia, the morbid process, not yet exhausted in one lung, continuing in the other. It is generally in pneumonias involving a very large extent of one lung that you see a new focus of inflammation developed on the opposite side, and it is a remarkable fact that the new inflammatory centre never attains, either in extent or in depth, the importance of the first. When resolution takes place—for double pneumonia is not necessarily mortal—it is always earlier and more rapid in the lung secondarily affected, than in the primary seat of the disease.

It will not do to attach too much importance either to the exacerbation of the fever or to the sensations of the patient, as interpreting the progress of the new pneumonic outbreak. If the secondary localization is effected during the most acute period of the disease, the thermometric curve, then high, will be slightly if at all modified.

When, however, the extension to the healthy lung takes place towards the decline of the primary pneumonia, one may observe a very high elevation of temperature, which it will not do to mistake for the critical exacerbation, precursor of the defervescence. In any event, there is no new chill, and rarely the patient complains of a pain in the side. It is, then, only by auscultation that the clinician can recognize the moment when the pneumonia becomes double. The blowing breathing and the bronchophony are often audible over the new seat of inflammation at the same time as the crepitant râle, and are earlier heard than during the first period of the primary phlegmasia.

One ought not, however, to conclude hastily in favor of a double pneumonia, as soon as certain anomalies are noted in the auscultation of the healthy lung. It is sometimes very difficult to determine if the blowing respiration which one hears is really a morbid sound linked to a new pulmonary lesion, or if it is only the echoing (so to speak), the propagation to the sound side of the tubular souffle of the diseased side. The recognition of crepitant râles should decide the question, but the souffle may be dry without crepitation; in this case, if percussion does not indicate an appreciable dullness over the seat of the bronchial respiration, the diagnosis of double pneumonia ought to be held in abeyance. When the patient lies on the sound side, it will not do to mistake for the râle of engorgement, the bruit of crepitation resulting from the expansion of the alveoli, a bruit which rarely fails to be heard at the moment when the patient is raised up in order to be auscultated. In fine, sub-crepitant râles are heard quite frequently in the posterior and inferior part of the sound lung. They indicate not a new centre of pneumonia but a simple condition of passive œdematous stasis.

Massive Pneumonia.

We adopt this name, proposed by Grancher, to designate that variety of pneumonia with obstruction of the bronchi by fibrinous exudation, described by Wintrich, Wiedemann, Wilks, etc. (See page 87.) The fibrinous obliteration of the bronchi gives the key to all the physical anomalies which characterize massive pneumonia and have caused it to merit the denomination of *pseudo-pleuritic*, given to it by Lépine. When the fibrinous bronchitis extends as high as the bifurcation, examination of the thorax gives the physical signs of pleurisy with effusion, and not those of pneumonia. Percussion gives an absolute dullness, "*tanquam percussi femoris*," (like percussing over the hips), with complete loss of the natural elasticity. The vocal vibrations are abolished under palpation. Auscultation reveals the

absence of every respiratory sound throughout the whole extent of the lung involved, in front as well as behind. Hence, absolute dullness, absence of vibration, of breathing sounds, of bronchophony, and of râles—such are the signs of massive pneumonia, and all the observations concur in this representation. If we add to this the frequent absence of all expectoration, and intensity of the dyspnœa, we can easily understand the mistake of most observers, who in presence of facts of this kind, have felt themselves under urgent obligation to immediately practise thoracentesis, convinced of the existence of an enormous effusion.

Such a mistake is, however, inadmissible, except when one finds himself face to face with massive pneumonia of the fourth or sixth day, that is to say, when already the bronchial obliteration is complete, and when one has before him a patient incapable of furnishing the least clue to the history of the case. The march of the disease is in fact the same in massive pneumonia as in fibrinous pneumonia generally. There is the same sudden onset with prolonged chill, followed by pain in the side, cough and dyspnœa. The observation of Henrot, and several other observations, prove that the ordinary physical signs of pneumonia exist in the period which precedes the bronchial obstruction.[1] In the case of Henrot, the crepitant râles, the tubular souffle and the exaggeration of vocal vibrations were appreciable during the greater part of the disease. It was not till the tenth day that the pseudo-pleuritic phenomena mentioned above were noticed. Moreover with the next day, the patient having expectorated seven bronchial concretions, the proper signs of pneumonia appeared anew.

The most important symptom at the moment of coagulation of the bronchial exudation, is the extreme intensity of the dyspnœa. The suffocation, the orthopnœa, the respiratory anguish, are at their height. The face is pinched and cyanosed; asphyxia appears imminent. Sometimes expectoration is nil, sometimes it exists, although scanty, with the characteristic aspect; in some instances patients have coughed up fibrinous débris and even casts of the bronchi. There is no need of insisting on the gravity of this form; death is the rule in massive pneumonia.

The diagnosis can only be made by taking into consideration the history of the case. If you concede an absolute value to the physical signs, confusion with pleurisy is impossible to be avoided; according as the rusty sputa are present or are wanting, the clinician will diagnosticate a vast pleuritic effusion, or a pleuro-pneumonia with exaggerated predominance of the pleural exudation. This is the error committed in the greater number of the published cases. As for the hypothesis of a pleuritic effusion, this cannot be admitted if one possesses any data respecting the march of the disease. Pleurisy does not begin suddenly by a violent and prolonged chill with a fever of 104° F. or 105°, and in any event, one never sees a case of pleurisy which may in five or six days determine the production of an effusion capable of filling the pleural cavity from top to bottom, and of effecting the abolition of every respiratory sound, normal or pathological. The confounding of massive pneumonia with pleuro-pneumonia is more easily explained. It is possible in fact (although exceptional), to see the pleurisy gain the ascendency over the pneumonia, and a quite abundant pleural effusion accompany the pulmonary hepatization. But however abundant may be this effusion, it cannot, in the space of several days, attain the degree necessary to produce the signs of massive

[1] Henrot, Notes in Clinical Medicine, Rheims, 1876.

pneumonia. The souffle and the œgophony are not wanting, and if the vesicular murmur may be abolished in the lower part of the thorax, it is never abolished throughout any great extent, and even in the event of excessive effusion, the œgophonic resonance of the voice is perceived, while it is wanting in cases of massive pneumonia.

Pneumonia with Bronchitis.

Around a focus of hepatization in pneumonia, are sometimes heard disseminated snoring and sibilant râles. These râles are only the index of the bronchial congestion which accompanies every pulmonary inflammation, and which constitutes a part of the pneumonic process, like the exudation on the surface of the pleura. But there exist other cases, and these are quite frequent, according to Grisolle, where true bronchitis precedes for a variable period the pneumonia. According to Grisolle, the proportion of pneumonias preceded by bronchitis is about one fourth. This is especially observed in the months of March and December. Sometimes it is an aged patient affected with ordinary bronchial catarrh, sometimes a younger subject taken with acute bronchitis *à frigore*. To this last category belong especially infants, so liable, during cold seasons, to inflammation of the bronchi. The snoring, sibilant, and mucous râles may be sufficiently intense and generalized to partly obscure the signs of pneumonia; sometimes also the rusty sputa may undergo modification, and even be lost in the muco-catarrhal expectoration. But in old catarrhal subjects, pneumonia has rather for effect to suppress the expectoration habitual to chronic bronchitis.

In any event, these pneumonias, grafted upon acute or chronic bronchitis, ought not to be confounded with broncho-pneumonia. They are distinguished by their march, which is always cyclical, by the sudden onset of the local and general symptoms, the limitation of the physical signs of induration to one side of the chest, and the typical defervescence. According to Jurgensen, the crisis is less clearly defined, and resolution is more slow and tedious, and is accompanied with a persistent febrile movement. This may be true in some cases, but cannot be regarded as the rule. The bronchitis may doubtless outlast the pneumonia, with a certain degree of remittent fever; but even in this event, in the clinical tracings representing the entire pyrexial march, the evolution of the pneumonia, from its beginning to its defervescence, is easily recognizable by its characteristic curve.

On the other hand, the existence of bronchial symptoms anterior to the onset of the pneumonia, does not authorize one in affirming the presence of an influenzal pneumonia. Apart from well marked epidemics of influenza like those of 1837 and 1884, one has no right to conclude in favor of the specific nature of the broncho-pulmonary symptoms, except when the bronchitis is accompanied with the general phenomena characteristic of influenza; intense lassitude, muscular pains in the limbs and loins, supra-orbital cephalalgia with nasal catarrh and hoarseness, febrile movement with evening exacerbations. If these phenomena are wanting, you have to do with an attack of intercurrent pneumonia supervening accidentally in an individual affected with bronchitis, (as may happen to an individual suffering from fracture of the leg,) and running its course without any anatomical or etiological connection with this bronchitis. At the most, one may admit that the bronchial inflammation furnishes a favorable soil for the culture of the pneumonic germ.

Pleuro-pneumonia and Pneumo-pleurisy.

Pleuro-pneumonia.—Every pneumonia which reaches the level of the pulmonary surface, determines the formation of pleural false membranes of soft and gelatinous consistence, which are more or less thick and more or less extensive. This exudative pleurisy is the constant rule in the pneumonias with infectant tendency. But by common consent the name of pleuro-pneumonia is given only to cases where the fibrino-serous exudation is sufficiently abundant to modify the signs peculiar to the pneumonia.

The frequency of pleuro-pneumonia varies, according to statistics, in the proportion of 12 to 15 per cent. In the greater number of cases the pneumonia is basal. Sometimes, however, the signs of pneumonia are perceived at the apex or middle part of the lung, and the pleurisy occupies the postero-inferior portions. The diagnosis is then easy. When the two lesions co-exist in the same region, the detection of absolute dullness with abolition of vocal vibrations and of the vesicular murmur does not warrant the diagnosis of pleuro-pneumonia. When a clear and well pronounced œgophony exists, doubt is no longer permissible. But in the contrary event, it is well to bear in mind the possibility of a fibrinous obstruction of the bronchi; the complete abolition of all respiratory murmur and the absence of œgophony do not belong to the pleurisy of pneumonia. The liquid effusion is in fact always in trifling quantity, even when the fibrinous exudation covers almost the whole of the lung, and Grisolle has been able justly to say that the quantity of liquid effused into the pleural cavity is in inverse ratio to the extent of the pneumonia. But these anatomical characters are precisely the conditions which favor essentially the production of the stethoscopic signs typical of the pleurisy, *i.e.* the blowing and acute quality of the respiration and the tremulous and œgophonic voice. When, therefore, there is complete silence during auscultation, the probabilities are in favor of bronchial obliteration. The probability becomes a certainty when this silence characterizes the entire extent of the lung. If the silence is limited to a circumscribed space, one should wait before pronouncing an opinion; sometimes the next day or even at the end of a few hours, the blowing breathing and the crepitant râles reappear in this point, thus testifying that the abolition of the respiratory bruit was due not to an abundant effusion but to a more ephemeral cause, without doubt to the limited and transient obliteration of some bronchial tube.

In pleuro-pneumonia it is ordinarily the case that the pneumonia obtains the ascendency over the pleurisy; the exception is when the pleurisy becomes the principal disease, while the pneumonia subsides. The latter alternative is, however, possible, and constitutes, as we shall see, the pneumo-pleurisy of Woillez.

Does the development of a pleuritic effusion affect in any way the prognosis of pneumonia? According to Laennec, the compression exercised by the liquid on the inflamed lung moderates the work of phlegmasia and arrests its extension. Grisolle, on the contrary, concludes from his observations that "in pleuro-pneumonia the pleuritic effusion appears neither to modify nor to circumscribe the inflammation. It cannot consequently modify the prognosis." When the pleurisy comes on during the acute period of pneumonia, it need not be an occasion of concern; sometimes it yields in several days, leaving the pulmonary inflammation to pursue its

course alone in the usual regular manner; sometimes it accompanies the pneumonia to defervescence, and the resorption of the pleural liquid takes place at the same time as the resorption of the alveolar exudation. Here also, according to Laennec, the pleuritic effusion hinders resolution, which is more protracted than in simple uncomplicated pneumonia.

When, on the other hand, the pleurisy comes on at the decline of the pneumonia, it presents different characters, and the prognosis is more sombre. Woillez proposes to describe facts of this kind apart, under the name of pneumo-pleurisies.

Pneumo-pleurisy.—In these cases, says Woillez, the pleurisy develops as a sequel to the pneumonia, at first as a latent disease, then as a grave pleurisy with rebellious effusion.

The onset is insidious; it is confounded with the resolution period of the pneumonia. Dullness and the tubular breathing are noticed, but these signs are attributed to the persistence of the pulmonary hepatization. The medical attendant neglects to auscultate the patient, believing him convalescent. It is not till after several days that the extension and the peculiar characteristics of the dullness fix the attention; all the signs of an abundant effusion are then found. According to Woillez this effusion is almost always purulent; he admits exceptions, however, and cites an example.

We must distinguish three varieties in these tardy pleurisies; the one, fibrino-serous, pursues its march independently on the termination of the pneumonia, with all the characters of simple primary pleurisy. The second variety, fibrino-purulent, seems to preserve something of the benignity proper to the original disease from which it was derived; if there is no interference on the part of the medical attendant it opens into the bronchi, and there discharges the contents of a pleural pus cavity; cicatrization and recovery following. In the case of an early evacuation of the pus by paracentesis, the purulent liquid does not seem to have a tendency to be reproduced, and cicatrization is rapid. Lastly, in the third variety, the purulent effusion, which is very abundant, fills the pleural sac, giving rise to the usual consequences of vast pus collections, and fatal termination is the rule; this last class answers especially to the facts witnessed by Woillez.

CHAPTER XIX.

INDIVIDUAL FORMS OF PNEUMONIA.

Frank Pneumonia of Childhood.

For long ages, the existence of pneumonia before puberty was denied on the authority of Hippocrates. At the beginning of this century, however, confusion of this affection with broncho-pneumonia exaggerated the frequency of inflammatory lesions of the lungs in young children. To-day, now that the distinction is made, it is admitted that frank pneumonia is rarer in childhood than in adult life. According to Ziemssen's statistics, which comprehend 186 cases of infantile fibrinous pneumonia, the disease is more common under six years of age; 117 cases in fact belong to the first six years of life, only 69 to the ten following years.[1]

We have said that pneumonia of the apex is frequent in children. According to Valleix it is more common than pneumonia of the base. Damaschino, out of 16 cases, notes 12 of the upper lobe to 4 of the lower. Likewise Verliac, out of 63 cases of pneumonia, found 34 of the apex and 29 of the base. But the more extensive statistics of Ziemssen, Rilliet and Barthez, which comprehend 263 cases of infantile pneumonia, give only 101 cases of pneumonia of the apex, against 162 of pneumonia of the base. It will not do, then, to insist too much on the relative frequency of localizations in children.

We do not intend here to give a complete history of the pneumonia of infancy and childhood, but only to indicate the most salient peculiarities. The abortive forms are common, but the duration may be as prolonged as in the adult, and attain nine or ten days, according to the extension of the local process, the march remaining cyclical, with characteristic defervescence. The existence of a previous bronchitis is not rare, and any one would mistake who should make a diagnosis of broncho-pneumonia from the sole fact that the pneumonia was superadded to an attack of bronchitis. The distinctness of the physical signs, the puffs of crepitant râles, with early and extensive tubular blowing, the situation of the lesions in the upper parts of the lung, the absence of every general affection predisposing to broncho-pneumonia, ought to suggest the existence of frank pneumonia with bronchitis, and it is easy to see how much more favorable a prognosis follows such a diagnosis.

The physical signs are the same in the child as in the adult, the absence of expectoration is the only particular worthy of note. These signs ought to be sought for with the greatest care, especially in the upper regions of the chest, in the supra-spinous fossæ, under the clavicle, and in the axilla, whenever an intense fever is all at once noticed in a young child, and which cannot be explained by the state of the throat or by a commencing eruptive fever.

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[1] Ziemssen, Pleuritis und Pneumonien im Kindersalter, Berlin, 1862.

The intensity of the general symptoms is the most interesting feature of the infantile pneumonias, and one which may provoke the most grave errors of diagnosis. The reverberation of the febrile perturbation on the nervous system in general, on the encephalon in particular, gives rise to those forms of pneumonia which Rilliez and Barthez have so well described under the name of cerebral pneumonia. These writers admit two varieties, the eclamptic form and the meningeal form.

The eclamptic form is the most common. The convulsions accompany the thermic elevations; they are only, as has been said, the exaggeration of the phenomenon of the chill. They manifest themselves here, as they are likely to do at the onset of every intense febrile affection in the young child. They are sometimes partial, limited to certain movements of the face and of the eyes, sometimes general, as in the great eclamptic seizure.

The meningeal form, more rare, is also much more deceptive.[1] The aspect of the child is absolutely that of a patient with brain fever. At

[1] "It is a well-known fact," says Dr. D. G. Brinton, "that, especially in children, pneumonia is apt to simulate meningitis. There is scarcely a text-book on the practice of medicine that does not make mention of this liability. But few cases, however, have been put on record to illustrate the possibility of such an error. For this reason the following case is very instructive, that Dr. Cadet de Gassicourt publishes in the *Revue Mens. des Malad. de l'Enfance*, January, 1885 :

"A boy, aged five, is suddenly attacked with vomiting and convulsions. The latter do not recur, but the former continues to the next day. Besides, a general lassitude is noted, headache, grinding of the teeth, and constipation make their appearance, and twenty-four hours later, somnolence, with gradual transition into coma, sets in. In this condition, the boy arrives at the hospital. There, besides the data already mentioned, divergent strabismus, and a mild degree of rigidity of the muscles of the neck, are plainly noticed. In consequence of these facts, and on account of the complete absence of any other indications, the diagnosis of meningitis is made, although the pulse was regular, not even slower (120 beats per minute), the abdomen not retracted, and no alteration of the pupillary reaction, present. The first symptom causing the suspicion of a pulmonary malady was very plain flapping of the alæ nasi, accompanied by a frequency of respiration amounting to forty per minute. The third day after his admission to the hospital there appeared a gradually increasing dullness on percussion at the angle of the left scapula, having first an extension of three cm., and on auscultation, the crepitant râle was plainly to be perceived. The first diagnosis was, therefore, dropped, and instead, that of acute lobar pneumonia recorded. This diagnosis seemed, also, to be confirmed during the next few days by a gradual increase—of degree as well as of extension—of the physical phenomena, until the eighth day, the whole part on the left side, anteriorly and posteriorly, gave a flat sound on percussion ; only below the clavicle in front of the chest, as well as at the back, a clear sound could be elicited.

"Less the local state of affairs than the general condition of the little patient soon produced, however, in G., the suspicion that the boy did not suffer from croupous pneumonia, but from that rare form of broncho-pneumonia to which G. has given the name of subacute pseudo-lobar broncho-pneumonia ; and it was especially the continuous lassitude which induced G. to this other change in his opinion.

"The tenth day the same physical signs as had been observed on the left side also made their appearance on the right, only not being quite so extensive there: and this fact served to confirm G. in his diagnosis. By increasing asphyxia, death set in two days later.

"The post-mortem examination showed a double-sided pleuritic effusion, besides pseudo-lobar left-sided broncho- pneumonia of the lower lobe, and disseminated pneumonia of the left upper lobe, and of the same lobe of the right lung.

"Besides the interesting diagnostic data, the case also contributes to medical literature one more proof of the infectious character of all these kinds of pneumonia."—Medical and Surgical Reporter, Aug. 22, 1885.—Tr.

first there is vomiting, constipation, cephalalgia, and delirium, which dominate the scene; then the child falls into a state of torpor with supineness, which may amount to a semi-comatose appearance, with immobility of the face and general insensibility; the belly is flat or excavated, the eyes are sometimes turned to one side, the pulse is sometimes unequal and retarded; the picture is, in fact, that of tuberculous meningitis.

But it is proved that these cerebral forms are especially observed in pneumonias of the apex, that is to say in cases where the physical signs are the slowest to manifest themselves, so that of necessity the diagnosis remains hesitating for several days. It is then the march of the accidents, and the history of the case, which should especially guide the medical attendant, and keep him from too hastily deciding in favor of tuberculous meningitis. He ought to be put on his guard by the suddenness of the onset of the cerebral phenomena; even in the acute sthenic form of tuberculous meningitis, certain intellectual modifications, peevish demeanor, headache, or some disturbances in the general health, precede the outbreak of the meningeal symptoms. Nothing like this exists in cerebral pneumonia. On the other hand, it is not usual to see tuberculous meningitis terminate in two or three days in somnolence or in coma. In fine, the elevated temperature of pneumonia is not ordinarily observed in meningitis. This anomalous train of symptoms having awakened the doubt, the attention will be directed toward the lungs by the acceleration of the respiration, the beating of the alæ nasi, sometimes by a slight cough. Auscultation, repeated after several days of uncertainty, will finally determine the true diagnosis.

Pneumonia of Old Age.

Pneumonia is the most dreaded scourge of old age, says Cruveilhier. From a pathological point of view, the lung is to the old person what the intestine is to the young child: it is par excellence the weak part, it is the open gate of entry to death. The pneumonia of old age nevertheless does not present any essential difference from that of adult life. It generally kills because the exhausted organism offers less resistance to morbid causes; but it may end in recovery if the subject is still robust, if he possesses the characteristics which constitute what is called a green old age. In these conditions the clinical evolution is identical with that of pneumonia of the adult; the mode of onset, the chill, the physical signs, the expectoration, the temperature, the critical defervescence, reproduce exactly the tableau of symptoms observed at earlier periods of life, as for instance between the ages of twenty and forty years. And it may be said that this frankly inflammatory evolution is the rule; all the authorities who have written on this subject are agreed on this point. If the pneumonia sometimes takes on a peculiar guise, the fact is exceptional, and can in no sense be due to the nature of the disease, which remains always the same, being explicable by the special conditions and properties of the soil in which the germ is developed. As in the case of the child, there may, as Gillette has remarked, be want of correspondence between the local lesion and the general symptoms; the latter are exalted to the maximum in pneumonia of childhood, while in old age reaction is reduced to the minimum. Hence those insidious forms of pneumonia called latent. On the other hand, the slowing of the circulation, the atheromatous state of the arterial system, the facility of cerebral alterations, explain the apoplectic

symptoms observed in so many cases. These, then, are the two special aspects recognizable in the pneumonia of advanced life.

Latent Pneumonia.—Examples of this kind are little seen except in the hospitals set apart for the diseases of old age, such as Bicêtre, or Salpêtrière. But if, as Charcot affirms, they cannot be called in question, they must certainly be regarded as exceptional. "The old ladies," say Hourmann and Dechambre,[1] "never complain even of malaise; nobody in their sleeping rooms, neither nurses nor waiters nor callers, notice any change in their position. They rise, make their beds, walk out, eat as usually, then feel a little fatigue, lean upon their bed and expire. We open the cadaver and find a large part of the pulmonary parenchyma in a state of suppuration." Prus even saw, at Bicêtre, an old man whom no one thought sick, fall dead in the court. The autopsy showed gray hepatization of the greater part of both lungs. Such facts do not seem to us possible except in the case of old people absolutely abandoned to themselves, and quite hebetated by age. The least examination would have revealed the pneumonia. They should, however, be regarded as examples of the feebleness, doubtless more real than apparent, of the constitutional reactions in old age.

Apoplectic Pneumonia.—Charcot and Vulpian were the first to call attention to those larvated forms of pneumonia, presenting themselves sometimes with the appearance of cerebral apoplexy with complete resolution and coma, sometimes under the aspect of a veritable hemiplegia with or without contracture of the paralyzed members. According to these two authorities, these phenomena do not correspond to any encephalic alteration, and they are reduced to the necessity of invoking an unusual kind of sympathetic action. Lépine, who has made a special study of these facts, has arrived at another interpretation. He explains the cerebral phenomena by the more or less generalized ischæmia of a hemisphere under the complex influence of the atheromatous alteration of the arteries, of the sanguineous inopexia proper to the pneumonia, and of the diminution of arterial tension consequent on enfeeblement of the heart: this ischæmia ordinarily being neither sufficiently pronounced nor sufficiently lasting to terminate in appreciable ramollissement. There are, in fact, not generally found in these pneumonic hemiplegias or apoplexies, either centres of softening or of hemorrhage. But the arteries are always very atheromatous, and sometimes one may notice a fibrinous clot obstructing one of the cerebral vessels. (Lépine).[2]

If the necrobiotic lesion of the nervous tissue is wanting it is generally because there was not time enough for the disintegration to become appreciable.

Apoplectic pneumonia, in fact, evolves rapidly. Sometimes the apoplectic stroke supervenes at the onset, accompanying the chill; sometimes the comatose and paralytic phenomena come on in the course of the pneumonia, towards the fourth or fifth day. In general, the patients have previously presented symptoms of cerebral anæmia, such as headache, dizziness, embarrassment of the speech, etc. When the fit takes place at the commencement, it follows almost immediately the initial chill; the patient smitten suddenly loses consciousness; the resolution is complete, there may be rotation of the head with conjugate deviation of the eyes. Then,

[1] Hourmann and Dechambre. Arch. Gén. de Méd., 1876, t. xii.

[2] Lépine, Soc. Biol., 1869, p. 349, and Pneumonic Hemiplegias, Thèse de Paris, 1870.

according to the gravity of the cerebral lesion, he recovers consciousness after a variable time; but he remains prostrated, hebetated; sometimes the ictus is followed by a complete or incomplete hemiplegia; sometimes the paralytic phenomena are wanting, and the shock with loss of consciousness is the only apoplectic symptom. The pneumonia evolves rapidly, and death arrives in several days.

When these accidents supervene in the course of pneumonia, you will see, at the end of three or four days, the old patients already delirious or in a sinking condition, or fall, more or less suddenly, into a comatose state followed or not by motor hemiplegia. The coma persists ordinarily till death, presenting nevertheless transient ameliorations and more or less marked aggravations.

The hemiplegic form is due to the localization of the ischæmia in the motor parts of the cerebrum, and this ischæmia may end in softening, as is proved by one of the observations of Lépine, and a case of Strauss. In the first case the ramollissement occupied the stratum intermedium of the left half of the pons; the patient had had right hemiplegia; in the other the lesion was seated in the form of a little spot of softening, scarcely seven millimetres in diameter, in the middle and internal part of the right lenticular nucleus, explaining the left hemiplegia noticed during the pneumonia.[1]

The apoplectic form without paralysis is doubtless amenable to the same interpretation. There is cerebral ischæmia, and foci of softening may be produced, but not occupying parts which preside over motility they do not determine hemiplegia. The observation of Lecorché and Talamon in fact demonstrates this association of phenomena. In an old man, sixty-five years of age, smitten with apoplexy at the commencement of an attack of pneumonia, and dead on the fourth day, without paralysis of the limbs, there were found at the autopsy very pronounced atheromatous alterations of the cerebral arteries, and besides two centres of recent softening, the one on the level of the second left frontal convolution, the other destroying the lobus quadratus of the same side.[2] Hence, whether the pneumonia be simply apoplectic or both apoplectic and hemiplegic, neither the one accident nor the other are reflex phenomena. These apoplectic and hemiplegic symptoms are linked to material lesions, more or less profound, and consequently more or less easily appreciable; lesions which pertain both to the state of the arteries in the aged subject, and the perturbation of the circulation provoked by the disease.

It will not do to confound these phenomena of cerebral origin with the cases of pneumonic paralysis reported by Macario of Nice.[3] These paralyses came on during the convalescence of pneumonia; they were of the same nature as the paralysis which succeeds acute diseases, and resembled diphtheritic paralysis. It must be admitted that facts of this kind are so rare that only the few observations published by Macario are on record.

Pneumonia of Drunkards.

Stokes was one of the first to call attention to the frequency of pneumonia with typhoid aspect in delirium tremens. "Of all the complica-

[1] Strauss, Note on a case of hemiplegia supervening in the course of Pneumonia. Revue Mens. de Méd. et Chir., 1877, p. 749.

[2] Lecorché and Talamon, Cerebral ramollissement in pneumonia. Études médicales, 1881, p, 412.

[3] Macario, Pneumonic paralysis. Bull. de ther., 1850.

tions which may cause the deaths of drunkards," he says, "inflammation of the lungs is the most frequent." Alcohol operates in these cases after the manner of a debilitating agent; by its slow and continuous action on the organism, it places the divers tissues in such a state of decay and dilapidation that they become incapable of all resistance to disease when it attacks them. Hence the quite special gravity of those pneumonias, which end almost invariably in death.

The inflammation often begins in the apex of the lung, but this localization cannot be regarded as the rule, for Stokes (significantly enough), has described the pneumonias of inebriates as pneumonias of the base. What characterizes these pneumonias is: 1. the extent of the lesions, which ordinarily occupy the greater part of one lung; 2. the rapid liquefaction of the exudation and its rapid transit to the stage of gray hepatization; 3. the adynamic state of the subject; 4. the delirium tremens. The alcoholic delirium existing in a more or less intense degree, is an essential fact. It appears from the first day of the pneumonia. Insomnia and agitation, some slight incoherence of ideas, are the first signs. You note a little trembling of the hands and tongue, then the delirium becomes more marked; it is ordinarily a busy delirium, and is characterized by terrifying hallucinations as of black objects. The agitation increases; the patients strive constantly to get out of bed; they continually throw off their bedclothes. At the same time the tongue becomes dry and black. The adynamic weakness is such that the patients when lifted up fall back like a dead weight upon their pillow. The pulse is of extreme frequency, the themometer oscillates between 104½° and 106° F.

It is in just such patients that you sometimes find at the autopsy fibrinous meningitis, scarcely suspected during life. It is easy to see how difficult it is, in the midst of the cerebral phenomena, to discern what belongs to *delirium sine materia* and what belongs to lesions of an inflammatory nature. Sometimes stiffness of the neck or of the limbs, or it may be a semi-comatose state, suddenly succeeding the agitation, suffices to indicate the new meningeal complication. As in infantile pneumonia, the cerebral phenomena obscure the pulmonary symptoms. The patients neither complain of cough nor of pain in the side; many do not expectorate. This obscuration may be so great, that the patient may even be sent to a lunatic asylum as a supposed case of simple delirium tremens, when in reality the delirium is symptomatic of an unrecognized pneumonia. The objective dyspnœa may attract attention in the direction of the lungs; but generally the physical and stethoscopic examination can alone enable the medical attendant to avoid the mistake.

All the pneumonias which supervene in individuals addicted to strong drink do not present this type of extreme gravity. It is evident that we must take into consideration the degree of alcoholic impregnation. The nervous manifestations may be limited to a little bustling delirium, to insomnia and a restlessness which exceeds the ordinary febrile agitation, but at any moment the accidents of delirium tremens are to be feared. And the knowledge of the intemperate habits of the patient ought always to make one reserved in the prognosis of a pneumonia which might otherwise be benign. In the matter of diagnosis, we are not concerned wholly with the difficulties caused by the intensity of the cerebral phenomena. It should certainly not be forgotten that under the ataxo-adynamic symptoms may be concealed other pulmonary lesions, such as tuberculosis with sub-acute march, which often puts an end to these old alcoholic subjects.

Pneumonia of the Diabetic.

Pneumonia may be observed in diabetes with all the characteristics of frank pneumonia, and may end in typical defervescence. Most authorities, however, have noted a less elevation of temperature, but it cannot be affirmed that the pneumonia of diabetes is apyretic. Certain cases are remarkable by the rapidity of the march; this is the fulminating pneumonia (*pneumonie foudroyante*) of Bouchardat. The patients succumb in twenty-four or forty-eight hours. These pneumonias are observed in diabetics exhausted by a prolonged flesh diet, or suddenly overcome by excessive fatigue consequent on a long walk or a journey. These are also, as is well known, the conditions which favor the development of that special state designated under the name of *acetonæmia*. And one might well ask if the gravity of the pneumonia is not in relation with this empoisonment, supervening at the same time. In fact, in the grave pneumonias of the diabetic, one often notices all the signs of *acetonæmia*. The intensity of the oppression is remarkable in these pneumonias, and cannot be explained solely by the local lesions. It must be accounted for in part by the alteration of the blood or some change in the medulla oblongata brought about by the acetone poisoning, of which dyspnœa is one of the principal symptoms. But the most characteristic feature is the special odor of acetone which is exaled in the breath of these pneumonic patients, an odor which recalls that of over-ripe apples or of chloroform. In fine the urine, when you turn into it a few drops of perchloride of iron solution, gives the reaction of acetone, taking the bright red color of Bourgogne wine. It is, then, difficult not to assign to acetonæmia a part in the special gravity of certain diabetic pneumonias.

As for the termination of these pneumonias by gangrene or by caseification we can only repeat what we have said in a previous paragraph. (See page 111.) The facts which have been published as examples of gangrene consecutive to pneumonic inflammation, may be interpreted as cases of primary pulmonary gangrene of the lobar form. Nevertheless, by reason of the tendency which phlegmasias in the glycosuric have to become complicated with mortification of the tissues, one may, although with some reserve, admit the possibility of this complication in the fibrinous pneumonia of diabetes. But with respect to caseification there is no room for the same reservation. From the day when Koch's bacilli were first noted in the sputa, the mistake was obvious; the pneumonia diagnosticated as fibrinous was a tuberculous inflammation.

Pneumonia of the Gouty.

May gout of itself determine an attack of fibrinous pneumonia, or does it offer only a special soil for the development of the pneumonic parasite, like diabetes and alcoholism? Is there a gouty peri-pneumonia, as our ancient predecessors called it, or are there only cases of pneumonia amongst the gouty?

Let us note, then, first of all, that whether we take into account the physical signs, or the general and febrile symptoms, the pneumonia which is observed in gout differs in nothing from ordinary pneumonia. It may run its course without anything to direct the attention to the joints, on any manifestation whatever of a gouty constitution. In a certain number of cases, however, you notice a sort of *balancing* between the gouty

articular phenomena and the pulmonary inflammation, and this is the capital argument of the assertors of gouty pneumonia. Sometimes,—and this applies to the majority of cases,—the pneumonia appears first, and after a normal march, about the sixth or seventh day, an attack of gout sets in; this is the *critical gout* of the ancients. Sometimes, on the other hand, there is a gouty paroxysm with swelling and pain in the great toe or in some other joint; then suddenly the joint symptoms disappear to give place to the usual signs of frank pneumonia. In still rarer cases, according to Lecorché, the ordinary articular crisis is replaced at the epoch when it generally appears,—in spring or fall—by a pneumonic crisis.

Are such facts as these sufficient to establish the gouty nature of these pneumonias? They are easily susceptible of another interpretation. In the first group of cases, the pneumonia behaves toward the gout as every intercurrent affection toward a chronic or constitutional disease. It excites, or awakens an acute attack of gout as a traumatism awakens a syphilitic manifestation, or as a cold in a malarial person provokes an attack of intermittent fever. When, on the contrary, the fit of gout seems to spend itself on the lung, when the articular crisis seems to give place to the pneumonia, in reality there is no such vicarious transference; the gouty paroxysm is suppressed by the pneumonia in virtue of the Hippocratic law which always holds good: *duobus laboribus simul abortis, gravior obscurat alterum.*[1] Moreover, in most cases the joint symptoms do not completely disappear; there is alteration but not delitescence. As for the third class of facts, when the pneumonia takes the place of a joint crisis of the spring or fall, it is easily explained on the theory that the intense fever of the pulmonary inflammation may have consumed and eliminated a considerable proportion of uric acid; an excess of this excrementitious product, the exciting cause of the gout, no longer existing in the blood, the paroxysm does not manifest itself.

We do not believe, then (and here we share the opinion of Lecorché),[2] that by the side of intercurrent pneumonias, supervening in the gouty, without any etiological participation of the gouty poison, there exists another series of pneumonias taking their origin directly from the uricæmia. Fibrinous pneumonia remains, in our belief, a specific affection, taking on variable clinical aspects and provoking divers reactions, according to the soil where it is developed, but always one in its causation and nature.

Pneumonia of Cardiac Subjects.

The pulmonary manifestations which are witnessed in the course of cardiac affections, generally are dependent either on blood stasis, or on embolism of the pulmonary arteries. Œdematous congestion with bronchitis and sanguineous effusion are the lesions which oftenest give rise to the symptoms observed. Frank pneumonia may nevertheless supervene in a cardiac patient, but from the point of view of the progress of the disease, as well as from that of diagnosis, we ought to distinguish the pneumonias which appear at the stationary period of diseases of the heart, (*period d'état*) from those of the period called asystolia.

When the heart affection is in its earlier stages, and this organ has preserved its energy and its regularity, whether the pathological condition

[1] "Of two morbid processes at the same time arising, the greater obscures the lesser."

[2] Lecorché; Theoretical and practical treatise on Gout. Paris, 1884, p. 324.

be aortic or mitral lesion, the prognosis of the pneumonia is not necessarily more unfavorable by reason of this complication. It would certainly seem from what we have said as to the rôle of the heart in the asphyxiating phenomena of the final scene, that insufficient valves or constricted orifices must favor the effects of venous engorgement of the heart, and conduce more rapidly to the paralytic dilatation. Generally, however, save perhaps a greater intensity of the dyspnœa, the pneumonia comports itself in a regular manner, and ends in recovery.[1] This hopeful prognosis appertains only to the earlier cases. If the cardiac disease has already lasted a long time, if the subject has passed the adult age, every reservation should be made. We have reason, in fact, to suppose in such patients the existence of a fatty alteration of the myocardium, and constriction of the nutrient arteries of this muscle by endarteritis, and we know by the observations of Jurgensen, the influence of these myocardiac lesions on the fatal termination of the pneumonia. As for the diagnosis, it does not present great difficulties. The bloody expectoration and the pain in the side noticed in an individual suffering from an affection of the heart, doubtless suggest a hæmoptoic infarctus. But the onset by a violent chill, the febrile elevation of the temperature, the clearness of the crepitant râle, do not allow one long to be mistaken.

The case is different when the pneumonia comes on during asystolia. Here diagnosis from pulmonary apoplexy is almost impossible. By reason of the general conditions in which the patient is placed, the temperature may scarcely exceed the normal. The only signs furnished by these pneumonias, which are essentially insidious, and are by so much the more obscure that the functional symptoms in large part disappear in the orthpnœa of the asystolia, are the expectoration and the stethoscopic phenomena. But the rusty sputa, by reason no doubt of the sanguineous engorgement of the entire lung, are almost completely formed of blood, and are easily confounded with those of pulmonary apoplexy by their color and their composition. As for the physical signs; crepitant râles, tubular souffle, bronchophony, they are also observed, although more rarely, it is true, in the case of extensive pulmonary infarctus and especially in such as provoke an inflammation of the pulmonary tissue and of the pleura in their vicinity. In the case, then, of a patient in the period of asystolia, the medical attendant can seldom say if he shall find at the autopsy a frank fibrinous pneumonia, or nodules of embolic infarction disseminated throughout an inflamed lung. It matters little to the patient which it may be, for frank pneumonia or infarctus with surrounding inflammation, have both alike a speedily fatal termination.

Pneumonia of the Cachectic.

The pneumonia of asystolic patients, by its absence of general reaction, makes part of the group of cachectic pneumonias. These pneumonias are in fact essentially characterized by apyrexia.

They are insidious in their local manifestations and a-febrile in their general manifestations. The apyrexia may be complete, as in the case observed by Wunderlich of a hæmatophilic patient, where, during the whole course of the disease, temperature remained sub-febrile. In general, however, there is a slight thermic elevation. Lépine, who gave to these pneumonias the name of *starvation pneumonias*, says that he has in several cases

[1] Grisolle.

seen the mercury in the thermometer rise to 100° F. and even to several tenths of a degree above, although the higher elevation was momentary, and the maximum of temperature was insufficient to cause one to suspect pneumonia. These pneumonias are, then, veritably latent; the pain in the side is scarcely felt, the rusty expectoration is wanting, the physical signs alone indicate the pulmonary lesion. These pneumonic attacks are observed in hæmophilic subjects (Wunderlich), in the insane, at the close of chronic diseases, in persons long deprived of sufficient food, in cancerous patients, and in particular, in cancer of the œsophagus. They are really frank fibrinous pneumonias, for the autopsy shows the characteristic lobar hepatization, and it is well known, moreover, that one can, even in a vigorous adult, by means of sufficient doses of chlorhydrate of kairin, cause a frank pneumonic inflammation to undergo evolution without the temperature exceeding 98½° F.[1] These ultimate pneumonias ought to be assimilated to the buccal thrush or to the pharyngeal diphtheria which so often set in, in like circumstances, to give the final blow to young cahectic patients.

[1] Filehne and Hallopeau.

CHAPTER XX.

DIVERS SPECIFIC PNEUMONIAS.

Malarious Pneumonia.

Malarious pneumonia is the best defined type of pneumonias due to a specific agent other than the parasite of primary frank pneumonia. Intermittent fever is an infectious disease, whether one admits or not as demonstrated the existence of the bacillus malariæ described by Tommasi-Crudeli as the agent of infection. It is the pulmonary localization of this infection which constitutes marsh pneumonia, and the latter takes on all the ordinary characters of intermittent fever. This pneumonia, admitted by most physicians who have practised in malarial countries, has been made by Grisolle the subject of a study to which we can add nothing to-day. Grisolle repudiates the idea of a pulmonary inflammation complicating an attack of fever and ague; for him the pneumonia and the intermittent fever are two pathological states linked intimately together and proceeding from the same miasmatic cause.

The view that pneumonia of intermittent form is only one disease complicated by another, has been at different times maintained. Thus Colin denies the existence of pernicious malarial pneumonia. According to him, the pulmonary phenomena, sometimes so formidable, observed in malarial patients, should be regarded as true pneumonic attacks of an extreme gravity, and of rapid march, comparable by their symptoms and their prognosis to the pneumonias of the scorbutic or of drunkards.

Such an interpretation seems to us chiefly applicable to the form described under the name of remittent pneumonia, and of pernicious remittent pleuro-pneumonic fever. The local lesion evolves like a grave pneumonia, but the fever, while being continuous, presents from time to time, at regular intervals, exacerbations and remissions. Grisolle himself is quite inclined to explain in this way a great number of facts recorded under this name, and he remarks that in marshy localities it is common to see, in certain seasons, the intermittent element complicating a great number of prevalent affections and in some way grafting itself upon them. But in intermittent pneumonia proper, this simple adjunction of the malarial element to the pulmonary inflammation cannot be admitted. It is not only, in fact, the general symptoms which take on the intermittent type but the local process as well.

The disease may begin in two different ways. Sometimes it is the febrile element which dominates the situation; the medical attendant thinks that he has before him a case of simple fever and ague, and it is not till the second attack that the pulmonary disease unmasks itself. Sometimes the local symptoms appear first with the ordinary signs of pneumonia, and it is only the subsequent march of the disease which reveals its paludal character.

The type of the fever is ordinarily tertian or quotidian. Between the paroxysms, the apyrexia is complete. To this apyretic period corresponds a remarkable amelioration of the local state. The dyspnœa, the cough, the pain disappear; the rusty sputa give place to mucous sputa; the physical signs themselves undergo notable amendment. May the latter disappear altogether? This is a controverted point. Certain authorities—Rousseau and Maillot for example—affirm it. Grisolle also admits it; at the same time he adds: "quite often during the apyrexia, physical exploration shows that the lung has not completely regained its normal properties. You may, for instance, still find a slight amount of large moist crepitation, or the respiration is harsher, or at least weaker than normal, or else you note a prolonged and even blowing expiration." The complete vanishing of the physical signs does not seem to us possible, except in cases where these signs are limited to the fine crepitation of the stage of engorgement. But when one perceives tubular blowing and the signs of red hepatization, it is evident from these indications that a sudden return of the lung to the normal state is not admissible. In an observation of Catteloup, the bronchial souffle was found to persist, despite the apyrexia, during the inter-paroxysmal period, which was two days, and the autopsy showed later red hepatization of the two lower thirds of the lung. What one must admit is, that the local process is arrested during the apyrexia, and does not progress till at the moment of the febrile attack, and that it is, in consequence, intimately linked to the cause which provokes the paroxysm. As for the physical signs, they can no more serve for a criterion here than in ordinary pneumonia. In order that a block of hepatization may undergo resolution and absorption, a certain lapse of time is always needed, and there is no reason to suppose that the physical conditions of liquefaction and resorption of the exudation are different in malarial pneumonia from what they are in ordinary pneumonia.

Of little consequence, then, is the persistence of the physical signs in the interval of the attacks; the essential fact is the parallel evolution of the local exacerbations and of the febrile paroxysms. If you succeed in extinguishing the paludal element by the specific treatment, by sulphate of quinine, the intermittent fever and the pneumonia disappear at the same time. If, on the other hand, the nature of the disease is misunderstood, the general and local phenomena are aggravated and the disease manifests the pernicious type. To the intensity of the paroxysms corresponds an exaggeration of the dyspnœa and of the pain; the pneumonia in these severe forms extends and gains both lungs, delirium comes on, and the patient succumbs in coma about the third or fourth attack.

The favorable influence of the quinine treatment ought to be held as the most evident proof of the specific nature of the pulmonary lesion till the day when certainty as to the causal agent of malaria, and the detection of this agent in the particular case, shall make the demonstration complete.

Influenzal Pneumonia.

The characters of these pneumonias ought especially to be studied in connection with the great epidemics of influenza. Sporadic influenza may also determine pneumonia, but the characteristics of the latter are little pronounced and sometimes quite difficult of recognition.

All authorities, as we have seen, have mentioned the frequency of pneumonic lesions in epidemics of influenza. In this respect the epidemic of Paris in 1837, as furnishing the most interesting materials for study, was the most remarkable. In one single term of service in the Hôtel Dieu, there were as many as 29 cases of pneumonia in the month of February; and during the same season, out of 300 deaths reported in this hospital, 80 were due to pneumonia.

Sometimes the influenza involves the lung from the very first; sometimes the pneumonia supervenes in the course of an influenza already characterized by multiple manifestations and by an intense bronchitis. But even in the first class of cases, the phenomena of nervous depression peculiar to influenza are never wanting, and manifest themselves under the form of prodromes. A general lassitude, a sensation of aching in all the limbs, and pains in the muscles, intense frontal and orbital cephalalgia and a feeling of depression and extreme prostration mark the invasion of the disease. At the same time, the medical attendant notes certain catarrhal symptoms of the upper air passages, such as snuffles, sore throat and hoarseness; after two or three days of this general malaise, the pulmonary localization manifests itself by the habitual signs more or less modified. In other cases the influenza declares itself by its proper symptoms; irregular and remittent fever of variable intensity, violent inflammation of the pituitary membrane, angina, laryngitis and general bronchitis with extreme dyspnœa, and it is in the course of these catarrhal phenomena that the pneumonia sets in, ordinarily about the sixth day, or on the ninth according to Landau.

This onset is far from being as well defined as in frank pneumonia. The chill is often wanting or is replaced by repeated shiverings, the pain in the side is confounded with the thoracic pains experienced by the patient. The expectoration is at first simply mucous and catarrhal, or muco-purulent, and in cases where the bronchitis is intense it may remain such during the whole course of the pneumonia. Generally, however, the sputa at the end of several days present the characters of pneumonic sputa. They become viscous, adherent, colored by the blood, and you find then in the midst of the purulent expectoration the brick-dust or even prune-juice sputa of lobar pneumonia.

The physical signs of the first period are likewise notably modified. One rarely hears the fine and dry crepitation bursting in puffs under the ear; the crepitant râle is larger and moister and often mingled with subcrepitant râles.

It is the signs of hepatization which furnish the best elements of diagnosis. The dullness, the augmentation of vocal vibrations, the tubular souffle and bronchophony are perceived from the second or third day. The hepatization may be limited and occupy indifferently the base, the apex or the middle portion. In general the tubular souffle extends rapidly and acquires an extreme intensity. Influenzal pneumonia is extensive and frequently it invades both lungs. More than half the pneumonias observed by Landau were double, —21 out of 40; Nonat also indicates the proportion of 4 out of 9.

The local phenomena are accompanied by a grave general condition explicable in part by the pulmonary lesions, but chiefly by the specific action of the poison of influenza. The intensity of the dyspnœa is especially remarkable, and is not always in relation with the lesions diagnosticated. The general aspect is that of a typhoid patient; the adynamic prostration is sometimes such that the patients do not seem to suffer.

The frequency of death varies according to the epidemics; in the epidemic of 1837 the mortality was very high, and death sometimes supervened in a manner almost as if by lightning. When recovery takes place the resolution of the pneumonia is always tedious; the bronchitis and the signs of induration persist for several weeks; convalescence is very long, and the appetite returns but slowly.

Erysipelatous Pneumonia.

This term has been used in three different senses. Certain medical writers thus designate those shifting pneumonias which we have before described under the head of migrating or ambulatory pneumonia; the serpiginous and irregular march of the disease resembling the cutaneous affection from which the name is taken. It is in this sense that Trousseau has employed it, having in view only the shifting character of the lesions. Others, under this term suggest a complete likeness between pneumonia and erysipelas. This was, as we have seen, one of the arguments of Traube in favor of the essential nature of pneumonic fever. Kussmaul makes the following parallel between the two affections:—"Etiologically pneumonia and erysipelas have this in common that they are both infectious and in some cases contagious. These two diseases appear simultaneously at certain periods of the year; both may develop secondarily in the course of infectious diseases. Symptomatically they are characterized by a high fever with sudden invasion, by a chill and a local pain, and by their cyclical march. Ordinarily the termination takes place by resolution, suppuration being exceptional. In both diseases the spleen is tumefied. In fine, contrarily to what one observes in other infectious diseases, recurrences are frequent in pneumonia as in erysipelas."

The value of the comparison instituted by Kussmaul is diminished by the sole fact of the differences of form and of properties which separate the parasite of pneumonia from that of erysipelas. But from an anatomical point of view, simply, the identity of the two affections cannot be maintained, for in cases of pneumonia truly erysipelatous, that is to say of erysipelas propagated to the lungs, the histological lesions differ from those of frank fibrinous pneumonia. These first two acceptations of the word *erysipelatous pneumonia* do not, then, seem to us to be legitimate; we can admit as erysipelatous only the pneumonia produced by propagation of erysipelas of the face, descending by the trachea and bronchi to the alveoli.

In general, the pulmonary symptoms appear on the decline of the facial erysipelas. They may be preceded or accompanied by the signs of erysipelas of the pharynx. Defervescence takes place, the disease seems terminated and the patient well, when a new febrile outbreak appears; there is no violent chill, and scarcely as much as a slight pain in the side is felt; auscultation indicates in a certain point of the lung a focus of crepitant râles. Then rapidly the signs of pulmonary hepatization extend; the general symptoms and the fever take on an abnormal intensity, and death arrives in the course of few days. Recovery has, nevertheless, been observed. (Stackler.) The clinical type of this variety of pneumonia is not, however, well established at present, and we cannot do better, in order to give an idea of the march of the disease, than to reproduce the summary of the case published by Strauss, which is one of the most carefully observed and best established on record. A young vigorous man, of intem-

perate habits, without grave previous disease, enters the hospital for an erysipelas of the face, which pursues its course without any marked peculiarity. The sixth day, the erysipelas having almost left the face, and the patient being convalescent, dysphagia set in, with bright redness of the pharynx, soft palate, tonsils, and tongue, (bucco-pharyngeal erysipelas). The tenth day, violent recrudescence of the fever, alteration of the features, slight pain in the right side without chill; not much cough. The medical attendant discovered pneumonia of the base of the right lung. The pneumonia extended with extreme rapidity. The sputa, at first viscous, of grayish brown color, took on the prune-juice aspect. In four days the right lung was invaded throughout its whole extent from base to apex; the temperature kept above 104° F. morning and evening. The adynamia was extreme, with meteorism, epistaxis, sub-icteric tint; and the fifth day after the onset of the pulmonary symptoms, the patient succumbed.

The diagnosis of this kind of pneumonia rests on the filiation and connection of the pulmonary symptoms with erysipelas of the face. The physical signs are those of ordinary lobar pneumonia. As for deciding whether you have to do with pseudo lobar broncho-pneumonia or with true lobar pneumonia, this is a question of purely histological interest. What is certain is that erysipelatous pneumonia is a specific and special pneumonia, distinct from frank pneumonia by its exudation, which is remarkable for its poorness in fibrine and its cellular richness, distinct from broncho-pneumonia by its localization in a single lung, and by its rapid spreading to the whole of the lung invaded, without any tendency towards lobular dissemination.

Pneumo-typhus.

Three distinct conditions have been described under this head: 1. frank pneumonia with general typhoid and adynamic aspect; 2. the hypostatic or other pneumonias, which supervene in the course of typhoid fever; 3. the pneumonias which sometimes mark the commencement of typhoid fever, and which may constitute the first and principal morbid determination. It is to this last category of facts only that we reserve the name of typhoid pneumonia, or pneumo-typhus. To avoid all confusion, the epithet typhoid, as we have already said, ought to be withheld from pneumonias of the first group: the typhoid condition is not, moreover, constant in those pneumonias which are especially characterized by their tendency to general infection of the economy. As for pneumonias of the second group, they are only complications of typhoid fever which do not merit a special designation.

Dietl, in 1855, was one of the first to describe pneumo-typhus in the sense of typhoid fever with primary pulmonary localization. "There are," says he, "certain typhic patients who from the very first present the signs and symptoms of pneumonia, as if the morbid intestinal determinations were transferred in part or in whole to the lungs."[1] Kremer also admitted, several years later, a primary pneumo-typhus and a secondary pneumo-typhus, according as the pulmonary symptoms precede or follow the appearance of the intestinal lesions. In primary pneumo-typhus, the intestinal symptoms may even be wanting, and at the autopsy none of the lesions of Peyer's patches may be found.[2] This is, moreover, the opinion

[1] Dietl, Weiner Wochens., 1855.
[2] Kremer, Canstatt's Jahresber., 1863.

of Griesinger, who describes, under the name of pneumo-typhus, the localization from the first and singly of the typhoid poison in the lungs. In the epidemic of pneumo-typhus observed by Gehrardt in 1875, the disease commenced by a frank pneumonia, then appeared the ordinary symptoms of typhoid fever, tumefaction of the spleen, lenticular rose spots, diarrhœa, and tympanism of the abdomen. Facts of this kind have been mentioned by Homolle according to Potain,[1] by Lépine,[2] and by Galissard of Marignac.[3]

Thus understood, the diagnosis of these typhoid pneumonias is extremely difficult. To differentiate them from adynamic pneumonias complicated with typhoid symptoms is almost impossible, apart from the existence of a clearly established epidemic. The physical signs of pneumonia in both cases are the same; the swelling of the spleen, the diarrhœa, the albuminuria, are observed in both diseases. The rose spots have a great value, but are there not typhoid fevers where these spots are completely wanting? The autopsy itself cannot always decide the question. In fact, in the infectant pneumonias with typhoid condition, Peyer's patches may be tumefied, as in every infectious disease; on the other hand, in pneumo-typhus, if we admit, with Griesinger and Kremer, one sole localization in the lung, it is possible to find Peyer's patches sound.

Under these circumstances till the day when we shall be able to find in the study of the micro-organisms proper to the two diseases the sure means of differentiating them, it will be prudent, from a purely clinical standpoint, to describe as pneumo-typhoid only the pneumonias immediately followed or accompanied by the symptoms characteristic of typhoid fever: the notion of a localization, absolutely single and isolated, of the typhoid microbe in the lung, being held in reserve, as it should be, for want of a demonstration which in the present state of science is impossible.

The typical pneumo-typhus may be thus described; the disease begins by the ordinary symptoms of lobar pneumonia, preceded by prodromes more or less pronounced; about the sixth or seventh day, at the epoch when defervescence takes place in frank pneumonia, the fever persists without remission, although the physical signs of the hepatization indicate its resolution; the abdominal symptoms are well delineated; the egg-yolk diarrhœa, the gurgling in the iliac fossa, the meteorism are manifest, the lenticular rose spots appear, and thenceforth the disease evolves as an ordinary typhoid fever with duration more or less prolonged.

As for the pneumonias which supervene in the course of typhoid fever, they cannot be confounded with pneumo-typhus. They come on towards the close of the second week, in those cases which the predominance of the symptoms of bronchitis and of pulmonary congestion has caused to be designated under the name of typhoid fever of thoracic form. The lesions of broncho-pneumonia, splenization and lobular hepatization combine with the hypostatic congestion, consequence of the dorsal decubitus, the adynamia, and the cardiac debility, to give rise to the signs of pneumonia. In the midst of sibilant and sub-crepitant râles, you hear, sometimes on one side, sometimes in both lungs, generally in the postero-inferior regions, a more or less intense bronchial souffle accompanied with bronchophony and with dullness. When the souffle is extensive, it corresponds to a pseudo-lobar hepatization. (Destais.) According to some

[1] Homolle, Rev. des Sc. Méd., t. x.
[2] Lépine, Rev. Mens. de Méd. et de Chir., 1878.
[3] Galissard de Marignac, Th. Paris, 1881.

facts reported by Marignac, one may observe a genuine lobar fibrinous pneumonia. It remains to determine if this fibrinous pneumonia is the direct result of the typhous process itself, like pseudo-lobar broncho-pneumonia, or if it be not rather a complication, or intercurrent affection, grafted on the typhoid fever.

Rheumatic Pneumonia.

In France rheumatismal pneumonia is considered as exceptional. Grisolle seems to regard as examples of this kind only cases where the invasion of the pneumonia coincides with diminution or sudden cessation of the articular pains. Looked at in this light, rheumatismal pneumonia is assuredly rare. Grisolle cites only four instances, of which two were observed by him, another by Andral, and a fourth by Dalmas. Another observation is recorded by Besnier in the thesis of Fernet. Lépine reports still another, a child of eleven years, a patient of Dr. Kobryner. In Andral's case, a man aged twenty-five years, affected with acute febrile articular rheumatism, remarked a sudden disappearance of the pains, of which the joints were the seat, at the same time that a considerable oppression supervened, accompanied soon by a dry cough; the third day the patient expectorated characteristic rusty sputa, and auscultation revealed pneumonia of the right upper lobe.

But this disappearance of the joint symptoms does not seem to us to be at all necessary. If in the course of an intense acute rheumatism one sees set in the signs of pneumonia, there is no valid reason for refusing to admit the rheumatic nature of this pulmonary inflammation. The English physicians evidently do not hesitate to do this, for they regard pneumonia as a frequent complication of rheumatism. In a careful statement of 246 cases of rheumatism, Fuller noted 28 intercurrent pneumonias, *i.e.*, a proportion of 1 in 9. Without giving any figures, Todd declares that pneumonia is often met in connection with rheumatic fever. It may precede, follow, or accompany the joint symptoms. These pneumonias ordinarily occur in cases of intense rheumatic fever complicated with endo-pericarditis and pleurisy.

According to Starges, they are characterized by the rapidity and suddenness of their evolution; quite abruptly a more or less extensive hepatization invades one, then both lungs. The gravity of these pneumonias is proportional to the extension of the pulmonary lesion, but it is especially in relation with the importance of the co-existing endocardial or pericardial lesions. In a case published by Raymond, the pulmonary phenomena, complicating a grave rheumatism, terminated by symptoms of cerebral rheumatism ending in death. At the autopsy the lesions were those of disseminated broncho-pneumonia, a fact which differentiates this case from ordinary rheumatic pneumonia where the physical signs observed are those of lobar pneumonia.

CHAPTER XXI.

BRONCHO-PNEUMONIA.

It is almost impossible to give a complete representation of the above disease in any formal description of symptoms, or to select clinical types which exactly correspond to the reality, so mobile and variable is it, whether we consider the malady in its general manifestations, or study it in the same subject. But what is especially variable, on account of the mode of dissemination and confluence of the lesions, is the physical signs. The functional and general phenomena remain the same, presenting only certain differences in their degree of intensity according to the extent of the disease. We may, then, comprehend them under one general and common description. In studying the physical signs we shall arrange the facts in two groups, which seem to us to correspond with sufficient exactness to what one observes at the bedside; the broncho-pneumonias where the signs of bronchitis predominate; the broncho-pneumonias where the signs of pneumonia predominate.

Modes of Invasion.

We have especially in view in our general description, the broncho-pneumonia of children; but the representation, with some few shades of difference, is the same in the adult and in the old man. The onset of broncho-pneumonia is not ordinarily easy to detect, whether the disease succeeds to the grave phenomena of suffocative bronchitis, or whether it comes on insidiously in the course of ordinary or specific bronchitis. There is neither the pain in the side nor the characteristic expectoration. Auscultation, as we shall see, gives only very vague information. The functional symptoms are the most important; the sudden augmentation of the dyspnœa (see p. 32), the peculiar character of expiration (expiratory respiration) on the one hand, the elevation of temperature on the other, with the extreme frequency of the pulse, ought immediately to suggest the idea of broncho-pneumonia; to this should be added the unusual agitation of the child, the injection of the countenance and the difficulty of dorsal decubitus. The chill of the invasion is wanting; sometimes general convulsions take its place. It is especially in measles that these eclamptic phenomena are observed. At all periods of measles, broncho-pneumonia menaces the infant, and may kill it at the very onset, under its subacute form; at the stationary period or during the decline it takes on a less rapid but always acute course; the eruption becomes pale, assumes a livid or violaceous hue, while the respiration and the pulse are accelerated, and the temperature rises.

When broncho-pneumonia comes on as a complication during the first few days which follow tracheotomy, divers peculiarities indicate the onset of the pulmonary inflammation. The canula becomes dry, and presents a dusky livid appearance within; the mucosities which

were flowing out by the wound are suppressed; the air of respiration in passing through the dry canula, takes that harsh sharp character which Trousseau designated under the name of *bruit serratique,* (stridulous bruit).

In whooping-cough the sudden suppression or attenuation of the convulsive paroxysms, coinciding with an intense febrile movement generally, indicates the onset of broncho-pneumonia.

Functional Troubles and Symptoms.

The disease being once established, the most important phenomena are those which concern respiration, the pulse and the temperature, and these require to be first studied.

Respiration.—The respiratory embarrassment is the capital symptom of broncho-pneumonia. The dyspnœa is at the same time characterized by acceleration of the respiratory movements, and by disturbance of the rhythm. The number of inspirations rises rapidly to 30, 40, and 50 per minute, and even more. In the terminal period one may reckon as many as 70 or 80 respirations. The dyspnœa may be aggravated at times under the form of attacks of suffocation.

The type of the respiration is modified. Not only the muscles of the upper part of the thorax, but the diaphragm and abdominal muscles contract with energy. At each inspiration the diaphragm draws in the costal cartilages and causes a sort of sudden sinking in of the inferior part of the sternum; at each expiration the abdominal viscera are in a manner aspirated into the thoracic cavity, and it is by this last movement that the respiratory cycle seems to commence. This is what is called the "expiratory respiration." (Bouchut.) "The series of movements," says Damaschino, "seems to begin by the expiration, which is performed suddenly and during which the abdominal viscera seem to enter the thorax. To this violent expiration succeeds immediately a powerful and short inspiration, during which the diaphragm again forces back the abdominal viscera at the same time that it determines a profound costal depression." Then there is quite a long pause, then the series recommences by a new and sudden expiration. Each respiratory movement thus commences by an expiratory shock, accompanied by a sort of play which is characteristic (Roger and Damaschino) and which sometimes enables one to make the diagnosis at a distance.

The aspect of the young patients then expresses extreme anguish; with lips and cheeks cyanosed and bluish, and eyes prominent, they maintain a sitting posture, hanging, it may be, for support, upon the bed posts, or the frame of their cot, thus struggling against the asphyxia, till at last exhausted they fall back on their pillow and give up the contest.

This peculiar aspect only belongs to the initial periods; if the disease is prolonged, prostration and sinking succeed this agitation. Although all the signs indicate an aggravation of the local state, the child seems to habituate itself at length to this sub-asphyxiating condition; the dyspnœa is no longer manifested except by the beating of the alæ nasi and the epigastric depression. The patient remains lying on the side, with pale, livid countenance, eyes shut, and in a state of somnolence and collapse, which continues till death ends the scene.

Pulse.—The pulse is accelerated at the same time as the respiration; one may count 130, 150, 180 pulsations a minute. At first sufficiently

strong and heaving, it becomes soft, small, contracted; in proportion as the asphyxia progresses the number of pulsations increases, soon it is almost impossible to count them. This excessive rapidity and this feebleness of the pulse ordinarily coincide with the formation of thrombi in the right heart.

Temperature.—The thermic curve of broncho-pneumonia is essentially irregular. It oscillates between 102° and 104° F. with morning remissions more or less marked and evening exacerbations. But now and then you note sudden ascensions, which make themselves apparent in the morning as well as in the evening.

These febrile exacerbations, according to Cadet de Gassicourt, correspond to local exacerbations of pulmonary congestion; they indicate the formation of new pneumonic foci. When the disease terminates in recovery, the fever progressively falls, but in a slow and tedious manner, and the curve is as irregular as during the active period: the temperature remains oscillating in the neighborhood of 103° F. and does not become definitely normal till after a time, sometimes very long. When the termination is fatal, in the very acute forms, the temperature remains very high, and attains the figure of 104° or 106° F. at the moment of death. In the protracted cases the thermometer no longer furnishes any certain data as to the state of the lungs. The first febrile outburst being over, you note the most variable temperatures, and one may even find at the autopsy extensive broncho-pneumonias with numerous nodules of gray hepatization, where for a long time the thermometer did not exceed 102° or 103° F.

The Cough.—The cough in the first periods is frequent, short, sometimes soft; it may also be paroxysmal and painful. It becomes more infrequent in proportion as the disease progresses. It is not accompanied by expectoration. The little patients swallow their sputa the moment they arrive in the mouth.

The other functional troubles offer little interest. The diarrhœa is frequent, as in the greater part of infantile diseases. The urine is scanty, high colored and charged with urates; it may be albuminous, especially in diphtheria. Convulsions, which sometimes mark the onset of broncho-pneumonia, may also appear in the course of the disease. They are then generally of fatal prognostic import.

Broncho-pneumonia with Signs of Bronchitis Predominating.

This clinical form corresponds, in a general way, to the facts described by Legendre and Bailly under the name of bronchitis with fœtal state and to the two anatomical types which we have adopted: generalized broncho-pneumonia and disseminated broncho-pneumonia. It is the physical signs of the bronchitis which predominate; if the tubular souffle appears it is in an intermittent, transient manner, soon shifting or else disappearing. But it should be known that the physical signs have only a relative value in the appreciation of the real lesions of the lungs. It is impossible to discern during the entire duration of the disease, aught but the signs of general bronchitis, and nevertheless at the autopsy numerous disseminated nodules of red or gray hepatization are found. From this point of view, one ought to accord much more importance to the duration of the broncho-pneumonia. If the pulmonary phenomena date back a fortnight, for a stronger reason if they are of earlier date, whatever may be the physical

signs furnished by auscultation, one may be assured that there exist lobules in red or gray hepatization, and generally in the depths of the parenchyma.

This being settled, what next are the different signs revealed by physical examination of the thorax? We can get little help here from either inspection or palpation. Percussion itself furnishes no information to aid diagnosis. The differences as to quality of pulmonary sound are insignificant, difficult to appreciate, *à fortiori*, to interpret. It is the stethoscopic phenomena which should chiefly engage the attention.

These stethoscopic signs are the râles of bronchitis, the obscurity of the vesicular murmur and certain soft and mobile souffles. The râles of bronchitis are the first signs perceived, they may be the only signs heard during the entire course of the disease.

The snoring and sibilant râles indicate only bronchitis of the large bronchi; they are heard in the upper parts of the chest, have no great value, and may, moreover, disappear shortly or be lacking altogether. The characteristic râle of the form of broncho-pneumonia which we are studying is the sub-crepitant, or fine *bubbling râle*. The abundance of these râles indicates the extent of the bronchi affected; their persistence in the same spots, coinciding with the functional and general symptoms which we have enumerated, denotes necessarily a lesion of the pulmonary parenchyma. This lesion will be atelectasis, with splenization or hepatization, more or less advanced, according to the degree of persistence of the bubbling râles.

The sub-crepitant râle is perceived, with its proper characters, in the posterior and inferior parts of both lungs. It is accompanied by a marked feebleness of the vesicular murmur. So many causes conspire to drown the vesicular murmur—the collapse of the alveoli, their dilatation by the emphysema, their obstruction to a greater or less extent by the lesions of splenization—that we cannot make much account of this sign. The vesicular murmur, enfeebled throughout the greater part of the lungs, may be completely abolished in a limited point, and this respiratory silence well noted in a circumscribed space in the thorax, reveals atelectasis of the subjacent pulmonary region. This atelectasis seems to us to explain another sign which all the authorities have noticed in broncho-pneumonia, but on which Cadet de Gassicourt has particularly insisted in his clinical Treatise on the Diseases of Children; we refer to the soft, erratic, variable souffle, of light intensity, which appears sometimes in one point of the thorax, sometimes in another, disappearing in the morning to reappear in the evening. Cadet de Gassicourt explains it by the mobile and fleeting congestions occurring in different points of the lungs, sometimes around nodules of hepatization, sometimes at a distance. It is possible that these active congestions exist, although a passive and fixed congestion seems rather to be the fact of broncho-pneumonia. But may a simple capillary congestion determine a souffle? The mobile element of broncho-pneumonia—is it not more correctly the atelectasis, the alveolar collapsus, which is susceptible of increase or diminution according to the displacements of the muco-purulent plug which obstructs the bronchial tube? And is not the condensation of the pulmonary tissue by the sinking in of the alveoli more likely to produce the blowing resonance of respiration, than the sanguineous engorgement of the vessels?

However this may be, these soft and mobile souffles, detected sometimes

at the root of the bronchi, sometimes in a circumscribed region of the chest, constitute, with the fixed and persistent bubbling râle, the symptomatology proper of broncho-pneumonias with bronchial predominance. We shall return further on to this clinical form when we come to consider its march.

Broncho-pneumonia with Pulmonary Predominance.

Anatomically the confluence of the nodules of broncho-pneumonia gives the key to the physical signs noted in the second clinical variety. According as the confluence is more or less pronounced, the signs bear resemblance to those of frank lobar pneumonia. Pseudo lobar broncho-pneumonia is the highest term of the series.

Both lungs may be affected in the same manner, the pulmonary consolidation being seated in the two lower lobes; but oftener the pneumonic form predominates in one lung, the bronchial form in the other.

The physical signs of bronchitis of the large and of the medium bronchi, exist at first alone; the fine bubbling râles in limited or more or less extensive foci, are perceived for a certain time; they may, however, be wanting. Then sometimes rapidly—almost at the very onset—sometimes, however, progressively, after a prolonged and variable period of phenomena purely bronchial, a focus of hepatization declares itself. This focus, at first limited, spreads rapidly and may invade an entire lobe. The signs are those of every pulmonary induration, save the augmentation of vocal vibrations, which cannot be perceived, except in children, toward the period of maturity. The first few days, percussion indicates only a slight degree of dullness, this dullness, however, soon becoming well marked. Respiration is at first simply blowing, accompanied by a few fixed râles during the violent fits of coughing. Then rapidly the souffle becomes rude and bronchial, sub-crepitant and even crepitant râles break under the ear with a dry timbre; the voice and the outcry have a noisy bronchophonic resonance, with a harsh sharp tone which rends the ear. The intensity and fixedness of the souffle, then, characterize the pneumonic form and indicate a confluent and extensive lobular hepatization. When death comes on rapidly the lesion does not go beyond red hepatization; when the disease is prolonged, the sharp and piercing souffle gives to the fixed bubbling râles a metallic tone; you often get the impression of a sort of cavernous râle, or of a veritable gurgling. According to Rilliet and Barthez, these signs indicate the existence of pulmonary excavations; this is, however, not always the case, but the autopsy generally reveals purulent capillary bronchitis with extensive gray hepatization.

March and Terminations.

Whatever may be the stethoscopic signs furnished by the disease, whether there may be predominance of the signs of bronchitis or of hepatization, cases of broncho-pneumonia, from the point of view of their evolution and duration, may be classed in the three following categories: I. Broncho-pneumonia with rapid march; II. Broncho-pneumonia with acute march—this is the ordinary form; III. Broncho-pneumonia with slow march.

Rapid Form.—The symptoms and signs are those of suffocative

catarrh. In extreme cases the march may be veritably fulminant. Death comes on in two or three days, especially in young infants and old people. These cases are such as are described under the name of capillary bronchitis. (See p. 32.) But the lesions of the pulmonary parenchyma exist as in the other forms. Only the generalization of the inflammatory process is such that time is wanting for its complete evolution. It is the extent and not the profundity of the lesions which brings on the fatal termination. In other cases the rapidity of the march is somewhat less. The disease is prolonged for six, seven, eight days, always characterized by asphyxia and the signs of capillary bronchitis, and at the autopsy are found more or less extensive nodules of disseminated hepatization. In fine, in a third group of cases the sub-acute symptoms undergo amendment at the end of several days, and the broncho-pneumonia, after having begun as a suffocative catarrh, takes the march and aspect of the common acute form. Acute tuberculosis is sometimes observed as a sequel of measles, with the same characters as suffocative broncho-pneumonia. The diagnosis is generally quite impossible.

The Acute Form.—This is the ordinary type of broncho-pneumonia. The physical signs are sometimes those of persistent bubbling bronchitis with mobile souffles of slight intensity, sometimes those of extensive hepatization. But you find here, in the highest degree, that irregularity which is almost characteristic of the march,—those sudden exacerbations of the temperature and those variable remissions, those aggravations and transient ameliorations of the general condition. The disease continues thus for a fortnight or three weeks, and death supervenes with the phenomena of progressive asphyxia coincident with a state of semi-coma and collapsus. Recovery is, however, possible, but rare. The respiratory embarrassment diminishes at the same time that the general and febrile symptoms improve. The temperature falls with irregular oscillations. The aspect changes for the better; the little patients ask for food, to be allowed to get up; the signs of bronchitis and of induration diminish, become circumscribed and end by disappearing.

Slow Form.—This form is especially observed as a sequel of measles or whooping-cough, in rachitic or cachetic children. It may begin like the acute form, in a sudden or insidious manner. But what characterizes it is the prolonged duration of the disease; death does not come on till at the end of five or six weeks. Here the evolution of the pneumonic lesions has had time to be completed. Hence it is in this form that one finds at the autopsy nodules of hepatization at all their periods, with predominance of the lesions of gray hepatization. The physical signs are both those of bubbling bronchitis and of extensive induration. There is heard over both bases a sharp tubular souffle mingled with numerous fine or large sized sub-crepitant râles with a tone almost cavernous. The fever has the characters of hectic fever. The little patients are lethargic, somnolent, silent; the complexion is of a yellowish pallor, the countenance sometimes puffy; the limbs are emaciated, almost skeleton-like; the skin flabby; diarrhœa is habitual. Death is the consequence of this progressive exhaustion, and it is sometimes very difficult to decide whether the lesions which one discovers at the autopsy are those of simple broncho-pneumonia or of tuberculous phthisis. Besides these two terminations of broncho-pneumonia—death habitually and recovery exceptionally,—a third alternative is possible; the passage to a chronic state. Certain sub-acute anatomical

forms seem to establish the transition between the lesions of the acute and those of the chronic state. Dilatation of the bronchi is the principal characteristic of these chronic broncho-pneumonias, and differentiates them from chronic pneumonias of croupous origin. But we must limit ourselves to a simple mention of this termination, the study of the anatomical processes belonging rather to the history of chronic affections of the lung and of pulmonary scleroses.

PART III.

CHAPTER XXII.

GENERAL THERAPEUTICS OF THE BRONCHO-PULMONARY INFLAMMATIONS.

Treatment of Pneumonia.

The new notions which we have acquired respecting the origin and nature of pneumonia bring with them consequences the most unexpected, and indications the most subversive of all the doctrines, ancient or modern, which have till to-day directed the treatment of that true inflammatory affection of the lungs known as frank, lobar, fibrinous pneumonia. Clinical experience, delivered from every theoretical prepossession, and judging the question without party bias, has for more than a quarter of a century shown that there is no curative treatment for pneumonia; henceforth and forever the line of inquiry should be in the direction of medicaments which shall attenuate the action of parasites on the organism, while at the same time maintaining the vital forces so as to enable them successfully to struggle against the invading and destructive action of the micro-organisms; here, then, is the causal indication. It is true that all the methods of treatment have always had this end in view, without ever attaining it, for under the mask of causation, physicians were in reality pursuing only the mechanism, the secondary determining conditions, or rather the lesions of the disease, believing that they could stay the mischief by modifying or arresting the morbid processes undergoing development.

This has been the rallying point of all the dogmas which have dominated the history of pneumonia from the time of Hippocrates to our day, and which have imposed themselves on therapeutics, and there is no system of medication what has not had its origin in some preconceived notion of causation; the prevailing pathogeny has governed the practice.

Pathogenic, Dynamic and Natural Indications.

As the theory, such the method. In the actual state of science we may affirm that the notions which we have acquired authorize discussion respecting only the three following therapeutical procedures: 1. That which is applied, not to the parasite, but to its primordial effects, in so far as it provokes the fever and the degenerations of organs; this is the *pathogenic method*, which comprehends principally antipyretic measures. 2. The method which consists in supporting the strength of the patient, to enable him to eliminate the virulent principle and to repair its disorders; this is the dynamic method. 3. That method which we may call natural or expectant, because leaving all to nature; it is the refuge of all the hesitancy and all the

doubts which beset us, and its only justification is its provisional and make-shift character.

When we come to understand the mode of action and results of these methods, we shall be in condition to judge of the futility of the antiphlogistic and contra-stimulant modes of treatment by which the medical mind has so long been befogged.

Doctrine of Peri-pneumonic Fever. Antipyretic Medication.

The true Hippocratic doctrine considers pneumonia as the local manifestation or fixation of a general disease, or of an ill determined principle pervading all the organs and humors. After a long eclipse, the pneumonic-fever theory (which, by the way, has never ceased to have adherents at Montpellier,) found in Germany, thirty years ago, a vigorous defender in Traube; later still Jurgensen has rehabilitated and given a new *éclat* to this ancient conception of pneumonia. The pulmonary phlegmasia comprehends a dominant element, which in all cases precedes and produces the lesion, constitutes its gravity and regulates its march; I refer to the hyperthermia. In view of the dissensions which still prevail respecting the origin of the phlegmasia; not knowing how to reach the malady in its primordial cause; not daring to act in accordance with the rigorous precepts of the spoliative methods; fearing even more to remain passive spectators of those morbid scenes which characterize each day and each hour of the evolution of the pneumonia, modern physicians have directed their endeavors towards combating the principal symptom—the excessive thermogenesis—which may be said to be of itself capable of producing disintegration of the vital organs and of compromising life. Hence the antipyretic measures, destined to combat the fever in all its constituent elements—the heat and the troubled circulation—while maintaining the heart's power; now the fever, being the direct effect of the action of the microbe, it may truly be said that antipyretics accomplish a fundamental indication, the *pathogenetic*, in assailing this first result. This method sanctions the usage of digitalis, of quinine, which in certain pneumonias fulfils the specific indication; of the derivatives of quinine, especially of antipyrine;—the other antipyretics, such as cold baths, have not come into general use in the treatment of pneumonia.[1]

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[1] ["Unless I am greatly deceived," says Professor Grasset, "all the new conquests of the parasitic theories have introduced nothing very subversive into the treatment of pneumonia, nor have they yet very profoundly revolutionized that old chapter of classical therapeutics." "When any one shall have shown me by decisive experiments that mercury has an action against the microbe of syphilis which it has not against that of malarial fever, and *vice versa* for quinine, I shall be willing to admit that the new theories have not exactly revolutionized therapeutics, but have given it a new scientific basis, a rational explanation which we have been long looking for." . . . "Now, with regard to acute pneumonia, I do not believe that experimentation has revealed a single medicament which has any specially marked action antagonistic to the microbe of this disease, and with all his ability and practical sagacity as a clinical experimenter, M. Germain Sée will find himself constantly obliged to return to indications drawn simply from the exaggerated symptoms, complications, and state of the patient—matters which appertain to the traditional therapeutics, and which the new doctrines have in nothing revolutionized." —Semaine Médicale, June 10, 1885, p. 199.]

"The antipyretic medication," says Grasset (loc. cit., p. 199), "is a new application of the organicist tendencies which endeavor to find in a dominant symptom and in certain lesions derived from it, the enemy which others seek in the pulmonary inflammation itself, or in the fibrine of the blood, which others still

Doctrine of Forces. Dynamic Medications.

At the end of the last century, Brown formulated, as a part of his system, the doctrine that all the acute diseases are owing to want of stimu-

more recently have sought for in the microbe. . . The traditional Hippocratic doctrine ends in a quite different conclusion. Seeing always the general disease, its cyclical evolution, its tendency to crisis, it falls back upon the natural method, which has for its aim, not to struggle against the enemy, but to watch the organism, to direct it if it deviates from the normal, and even to bring it back to the normal when far out of the way, but which always counts on the organism itself to effect its recovery, as it has made its disease."

To these and other strictures of the professor of Montpellier, Prof. Sée replies:

"The adoption of the pneumonic-fever theory by the Germans (Traube, Jurgensen, etc.,) seemed for a time to give support to the peculiar views which your school has ever entertained in opposition to the organicist theories of the school of Paris. The discovery of the microbe of pneumonia has upset those former notions. Nor do I believe that the hypothesis of *zymases*, which you seem to prefer, is a more intelligible or probable theory.

"We have a tolerably clear idea of what a microbe is; we see it, we stain it, we cultivate it. The zymase theory seems to me to be a conception bordering on that of the vital force, which we are destined never to comprehend with much clearness. To struggle against the zymase seems to me a struggle of the same kind as Jacob's wrestling with the angel.

"I confess that, thus far, from the point of view of the pneumonic microbe, the results of the contest are of little importance. Hence it is that I have not counselled any anti-parasitic specific in the treatment of pneumonia. It is not as *anti-microbics*, nor even as *anti-zymasics* that I prescribe quinine and antipyrine, but simply as antipyretics. And I endeavor with them not only to combat the hyperthermia, but to fortify at the same time the cardio-vascular energy and to sustain the nervous and trophic forces in the struggle of the organism against the parasitic infection. And this is why I supplement them by alcohol, good food, etc. Since I cannot directly destroy the assailant, I furnish to the besieged the munitions which enable it to prolong its defense and to tire out the enemy. You object that this is not any revolution in the art of treating pneumonia. It is not the year 1789, I own, since Todd had before me defended the same ideas; but admit with me, at least, that the profession is not much in advance of the year 1830, since we see around us physicians who continue to bleed, to give emetics and contra-stimulants to their pneumonic patients under pretext of combating the disease."

With regard to some objections which Prof. Grasset had made relative to the author's view as to the tolerance of medicines, Prof. Sée remarks:

"I have said that the more profoundly the organism was attacked and modified by the disease, the larger the doses necessary of the remedy, and that a sort of intoxication was often requisite in order to obtain the effect desired. I have said that neither the want of absorption nor the want of elimination could explain the apparent tolerance of the remedy, and that the condition of this tolerance was the gravity of the disease. You protest against the word intoxication in virtue of the principle that toxic actions are in inverse ratio to therapeutic actions.

"I do not discuss the value of this axiom. I cannot, however, help from considering as toxic the dose of ten to fifteen grammes of tincture of digitalis, which a patient affected with delirium tremens would bear, and which would certainly kill a person with heart disease. You will add that a disease does not need to be grave in order to entail tolerance, and in this connection you cite paludism and syphilis. These instances do not seem to me to be conclusive. No one, I think, will dispute that the more intense the malarial attack, the better quinine is tolerated, and that pernicious fever claims larger doses of quinine than ordinary intermittent. As for syphilis, I cannot admit that the syphilitic patient supports mercury in doses that the healthy man could not tolerate.

"The syphilitic subject supports mercury so poorly that, at the time when this medicament was administered *larga manu*, the patient lost by this insane treatment his teeth and his maxillaries, without any tolerance, real or apparent. There are surely idiosyncrasies with respect to mercury; certain persons bear it in doses which others do not tolerate; but it is not necessary to be syphilitic for all this.

lus.[1] From this was deduced the necessity of arousing the languishing functions, of strengthening the organic forces during the entire evolution of the disease. This doctrine, which may be called true vitalism, had almost passed into oblivion when Todd, in the spirit of Brown's teaching, prescribed alcohol in large doses in the treatment of fevers. Clinical experience gave justification to this venturous practice, and experimental physiology still further confirmed its utility. This is the medication which is the most rational even at the present day, and for several reasons: 1. alcohol serves as an aliment, directly absorbable by the veins, without any previous metamorphosis; 2. it sustains the bodily forces, in preventing denutrition; 3. it also sustains the forces by its direct action on the nervo-motor system; 4. it acts as an anti-thermic and possibly as an anti-parasitic agent. Perhaps we should also class among dynamic medicaments the excitants of the skin, such as vesicants and blisters. If these have not the power to awaken vital action, they at least deserve honorable mention among the symptom-treating measures designed to assuage pain and relieve dyspnœa; it is true that even the possession of this humble property is not conceded by all authorities.

Naturist Doctrine,[2] and Expectancy.

Dissatisfied with the ancient doctrines and methods many modern practitioners went back to the *vis medicatrix natura* as the standard of every true therapeutical endeavor. From the moment that pneumonia came to be considered as a fever, and later, in accordance with modern discoveries, as an infectious disease, the fact was recognized that, after the fashion of a great number of parasitic affections, it undergoes a regular course, a cyclical march, and manifests marked tendencies towards a crisis; or in other words, the spontaneous elimination or destruction of the morbid principle.

Syphilis is a chronic disease, and medicinal tolerance has nothing in common with chronic affections; neither cancer nor tuberculosis, nor diseases of the heart create tolerance. When I speak of grave diseases I mean acute diseases, which bring to bear on the nervous system and on calorification, a profound excitant or depressant action.

"This necessity of large doses does not by any means imply that the therapeutic action (on a sick man) must be conceived differently from the physiological action on the well man. I do not see there any argument in favor of the difference of the mode of actions of medicaments in the state of health and in the state of disease; it is only a difference of dose; and the only conclusion to draw from the point of view of the phenomenon of tolerance, is that the sick nervous system is with more difficulty impressed by therapeutical agents than the healthy nervous system."

Prof. Sée concludes by the remark that, though differing in theory with his confrère of Montpellier, he agrees substantially with him in practice, a supporting treatment which he calls a *nourished expectancy* (*une expectation nourrie*), being the sum of attainment in the present state of therapeutics. (Semaine Médicale, 1885, p. 223.)—Tr.

[1] John Brown, founder of the Brunonian system, was born in 1735 in Berwickshire, England, studied medicine at the Edinburgh University, and became tutor to the children of the celebrated Dr. Cullen, and assistant in his university lectures. He afterwards commenced giving lectures himself upon a new system of medicine, according to which all diseases are divided into the sthenic, or those depending on an excess of excitement, and the asthenic, or those resulting from a deficiency of it; the former to be removed by debilitating medicines, as antimony, the latter by stimulants, such as wine and brandy. In 1779, Brown took his degree at the University of Edinburgh, and in 1780 he published his Elementa Medicinæ. Brown died in 1788.—Tr.

[2] Doctrine Naturiste (Fr.).

This is what takes place after a brief space of time which can generally be foretold; certain days, and especially odd days, being designated as critical. With thermometer in hand, Traube, who held to the doctrine of pneumonic fever, followed with rigorous care the disease through its entire evolution, from the initial chill to the day of defervescence or of lysis, of sudden or slow extinction of the febrile heat. The fever was then estimated by the products of combustion, the urea, the uric and phosphoric acids and phosphates of the urine, and it was thus that a definite meaning was given to the ancient doctrine of crises. But from all these studies bearing the stamp of scientific observation, there follows this supreme law, which dominates the pathology of acute diseases:—*the curative efforts of the organism are ever manifest and are all important;*—whence comes the irresistible inference that these curative efforts should be respected and favored. Expectancy is the natural outcome of these considerations, and it has been raised to the height of a dogma, being regarded as the true method to oppose to the meddlesome and active but blind and dangerous therapeutical systems of the ancient masters of medicine.[1]

Doctrines of Inflammation and of Irritation, Antiphlogistic Medication, Contra-stimulants.

There are certain old theories and methods which henceforth deserve mention only for their historic interest.

Organicism.—The discoveries of pathological anatomy, rendered ever memorable by the practical labors of Laennec, Andral and Cruveilhier,—the knowledge of the anatomical lesions of the inflamed lung, gave rise to the doctrine of organicism, which was based on the notion of the local state when e was derived the entire series of phenomena styled reactional. The only thing now to be done in view of the known local lesions, was to com-

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[1] The general doctrine set forth in this paragraph is well stated by the late Dr. Jacob Bigelow in his work on "Nature in Disease," published more than thirty years ago. The first discourse in that work, "on self-limited diseases," was delivered before the Massachusetts Medical Society twenty years previously, viz. in 1835. In this chapter, Dr. Bigelow shows that the deficiency of the healing art is not justly attributable to any want of sagacity or diligence on the part of the medical profession, but to the inherent difficulties of the case. He shows that certain morbid processes in the human body have a definite and necessary career, from which they are not to be diverted by any known agents. By *self-limited diseases* he understands diseases "which receive limits from their own nature and not from foreign influences, which, after they have obtained a foothold in the system, cannot in the present state of our knowledge be eradicated or abridged by art, but to which there is due a certain succession of processes to be completed in a certain time, which time and processes may vary with the constitution and condition of the patient, and may lead to death or to recovery, but are not known to be shortened or greatly changed by medical treatment." As examples of these self-limited diseases he enumerates the acute infectious diseases (exanthemata, typhus and typhoid fevers), acute rheumatism, and gonorrhœa, dysentery, and he is inclined to include pneumonia, though he stumbles at the fact that blood-letting in proper cases lessens the severity and danger of this disease! He concludes with the motto, *Nature duce ;* the physician in his estimation being, as Hippocrates taught, only the servant and minister of nature.

The progress of knowledge since the time when Bigelow wrote, has only given emphasis and confirmation to these views, so that the physician of to-day may sum up the conduct of the practitioner at the bedside of a patient ill with croupous pneumonia, as well as with acute infectious diseases generally, in these words of Jurgensen : "*Nature cures, and the only duty of the physician is to maintain life until the cure is effected.*"

bat this initial congestion and inflammation in order to get the better of the malady; the depletive treatment, general and local, was supposed to fulfil the leading indication. This was the end and aim of the venesections and the blood-lettings *loco dolenti;* the latter constituting the real novelty, as general bleeding had for centuries held an important place in the therapeutics of so-called peri-pneumonia or pleurisy.

Modern Chemistry.—By the side of the organicist doctrine was soon ranged the chemical doctrine, having equally blood-letting for its consequence. Instead of regarding the inflammation as a result of the hyperthermia, and the latter as due to the impression of cold, the phlogosis was considered from a more general point of view. In Germany and shortly after in France, where there never lack physicians to adopt and cry up every medical absurdity, especially one bearing a foreign stamp, the blood itself was accused of all the mischief associated with inflammation; this vital fluid was believed to be too strong in fibrine (hence the name hyperinosis), and for this reason in just the condition to deposit its excess of fibrine in the lungs; from this was deduced the principle that the main concern in the treatment of inflammatory diseases was to deprive the blood of its phlogogenous elements. This could be accomplished only by anti-phlogistics, that is to say by blood-letting practised to depletion; hence organicism and chemistry were found in agreement in sanctioning this detestable practice of debilitative spoliation.

Contra-stimulation.—As a sequel of these excesses in blood-letting there sprang up in Italy a doctrine which has made much noise and done much harm. Brown, as we have seen, laid down the principle that every febrile disease was due to a want of stimulus; Rasori taught the opposite —that in fact all febrile inflammatory diseases are the expression of a diathesis which is characterized by excess of stimulus. The Italian school found in France numerous partisans, the most illustrious of whom, Laennec, committed there perhaps his only mistake. The means of counteracting this excess of stimulus was by giving in large doses a medicament which we now recognize as a powerful debilitant—a paralyzer of the muscular system, including the heart; I refer to tartrate of antimony.

CHAPTER XXIII.

ANTIPYRETIC MEDICATION.

Enumeration of Antipyretics.

Digitalis.—The first clinicians who made a special study of febrile heat, Wunderlich and Traube, made trials of digitalis, and recognized the property which this medicament possesses of slowing the pulse while sustaining vascular tension and cardiac innervation.

Quinine.—The same effects and the same advantages were attributed to quinine, which was later exclusively reserved for pneumonias of malarial origin.

Antipyrine.—Quite recently the quinine series has been enriched by two new medicaments, whose action is more prompt while less specific than that of quinine; these are kairine, already essayed without great success, and antipyrine, which is just taking its place in the group of antipyretics. Here the series of antipyretics available in pneumonia really terminates, but there are other remedies which lower febrile heat, though less advantageous, and more or less contra-indicated in pulmonary inflammations.

Antithermic Medicines.[1]—The salicylates and salicylic acid are extolled as powerful antithermic medicaments, which they undoubtedly are; they are, however, without effect in raising the heart's power and are likely to depress it, often causing besides a peculiar kind of cardiac dyspnœa.

Cold Baths.—Cold baths, according to Jurgensen[2] and his followers, constitute the ideal antithermic treatment; happily this mode of treating pneumonia has never prevailed in France.

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[1] An *antithermic* medicine is one that counteracts *heat*. An *antipyretic* antagonizes *fever*, in all its essential elements. *Antipyretics* are therefore more properly *febrifuge* than *antithermics*, while, in the sense signified by the derivation of the words, all medicines that are capable of lowering abnormal heat are antithermic.—Trans.

[2] Jurgensen, acting on the principle that in pneumonia "the fever is the first point of attack for treatment," and that "the force of the disease is really broken when the fever abates," and in accordance with his proposition, *sine thermometro nulla therapia*, does indeed teach that it is proper to abstract heat directly, and does not hesitate to expose the patient to fresh air,—preferring even the presence of a draft in the sick room to bad air,—and in the case of strong persons, moderately ill, and with no complications, he even administers baths of cold water, as in typhoid fever, when the temperature in the rectum reaches 104° F. He does not, however, apply this treatment to all cases of pneumonia, and recognizes fully the fact that the duty of the physician involves prophylaxis against exhaustion of the heart, which may, in weakly persons, become overloaded, and soon paralyzed by the bath. In children he sometimes employs wet packings, a mode of practice also recommended by Niemeyer. The benefit which can be derived from ergot of rye, administered in pneumonia in doses that are safe, is very problematical. It has never been proved that this drug contracts the blood-vessels of an inflamed lung, and the instances recorded by Wycisk, Wells, Boggs, Hanfield-Jones, Yeaman, Duboué, Scearce, and Holmes, of recovery from pneumonia under the use of ergot, are simply examples of the *post hoc ergo propter hoc* fallacy, which has done so much mischief in therapeutics.—Trans.

Muscular Poisons.—We find by the side of antithermic drugs, certain true poisons of the heart, such as veratrum and veratrine, which, however much they have been used in America, have not come into vogue in this country.

Vascular Medicaments. Ergot.—We should lastly mention ergot of rye, the property of which in determining contraction of the blood-vessels is now well recognized. The Americans have employed it, according to Duboué of Pau; we essayed this medicament in one notable case in concert with Dr. Boggs, the patient making a good recovery.

End of Antipyretics.

The end which the physician seeks to attain by the administration of antipyretics (which, by the way, hardly merit this name, as will appear from the analytical examination of their individual effects) is to combat the hyperthermia always and everywhere. We are not concerned here with the treatment of a mere symptom, or of a simple unimportant incident of the disease; it is a struggle against the master phenomenon which dominates the morbid scene, without, however, being the real cause of the disease.

We are compelled to regard the fever as a real *consumption;* this is proved by the increased elimination of urea and of carbonic acid. The fever means, moreover, granulo-fatty degeneration of the organs, especially of the liver, the kidneys, the muscles, and the heart muscle as well as the others. Besides the fatty degeneration of the heart, the fever is also accountable for the state of depression and enfeeblement of this organ, concerning which Jurgensen has written so truthfully, although with erroneous practical conclusions. Fever signifies, moreover, increased frequency of the pulse and quickness of circulation, and especially dicrotism of the pulsations, which is the presage and sign of feebleness of the vascular walls; the fever constitutes finally the hyperthermic dyspnœa which has been so well studied by Richet.

Every antipyretic, in order to claim a place in this important series of medicaments, ought to fulfil divers indications pertaining to the different manifestations of the pyrexia. It is not sufficient that it shall lower the temperature, that it shall slow the pulse; it must also sustain the heart's action, otherwise it will fall into the category of muscular poisons, such as veratrum and antimony, against the use of which we shall be compelled earnestly to protest.

New Notions Respecting Hyperthermia.

Eighteen months ago, Unvericht published a treatise which passed unnoticed, on fever and the antipyretics; recently at the clinic of Königsburg, Naunyn treated the same subject from a critical and experimental standpoint. Both these writers took ground against the data generally admitted respecting the danger of hyperpyrexia and the utility of refrigerants. These researches may be summarized as follows:

1. The excessive thermogenesis is but a symptom; the frequency of the pulse, the dyspnœa, the cerebral phenomena, the troubles of nutrition, are independent of the temperature, as has been already indicated by Volkmann and Genzner; they may even be apparent when the condition is apyrexial (Fräntzel.) All these phenomena and the pyrexia are without doubt the direct effect of the virulent principle.

2. The hyperthermia is not the cause of death; hares enclosed in a tight box whose temperature is maintained at from 105° F. to 107°, and even 110° F., may live for two weeks; at a higher temperature death supervenes by reason of the rigidity of the striated muscles, including the heart.

But Charles Richet has shown that one may raise the temperature in dogs to 108° F. with impunity on condition that the dog shall not be muzzled; the animal is cooled by its lungs. The argument of Naunyn is, then, without value; his hares were subjected to heat in a furnace, the febricitant derives his excess of heat from his interior environment.

3. In the experiments detailed the organs reacted against the elevation of temperature very differently; the systemic heat, as well as that of the blood of the carotid, seemed to excite violently the respiratory centres without exercising the least effect on the centres of innervation of the heart and of the blood-vessels. (Fick.) These are the results of experiments in artificial calorification.

4. In the pathological state the phenomena are different. In certain febrile maladies, says Naunyn, the elevation of the temperature constitutes in most patients the measure of danger; this is what one sees especially in pneumonia and typhoid fever; he adds: "The thermometric observation is the most certain basis of prognosis in the pneumonic and typhoid patient;" with this proposition we agree.

5. In the infectious diseases the absolute degree and even the duration of the hyperthermia have not always the same value; recurrent fever raises the temperature to 107° F. without producing serious troubles in the other functions; typhoid fever may last eight days and longer with temperature of 105½° F. and even higher, without decidedly compromising life. This proves the specialty of action of the divers virulent principles, nothing more.

6. In the infectious diseases, it is the virulent agent which certainly determines the fever; the infection as it progresses influences the various functions so that the hyperthermia and the other associated morbid manifestations march in front (so to speak), without, however, always exactly coinciding.

7. In pneumonia it is not the fever which determines the weakness of the heart, and death by the heart, as Jurgensen believes. The mortal collapse is the result of a generalized infection whose foundation is in the lungs; this is what results clearly from the studies of Naunyn, and of which we have given the proofs.

Remarks.—These conclusions seem to me irrefutable; they fully justify our appreciation of antipyretics, by giving which, we thus combat the principal but not the only factor of danger, the hyperthermia. This medication also enables us to oppose the other effects of the parasitic infection, namely, the nervous, cardiac and respiratory troubles, of which it is, moreover, almost impossible to say whether they are under the direct domination of the fever or in strict dependence on the microbe.

The question being thus settled, we may undertake the practical study of the antipyretics in general and the special history of quinine, of digitalis, and of antipyrine.

CHAPTER XXIV.

GENERAL PRECEPTS OF PATHOLOGY. TOLERANCE OF MEDICINES.

The notion of the tolerance of medicaments in diseases was the outcome of the study of contra-stimulants, such as tartar emetic in large doses. It was remarked that the medicine does not take effect unless one succeeds in steering clear of its emeto-cathartic action, and in causing to be introduced, absorbed and *tolerated* the greatest quantity possible: it is then that it has its maximum of power to combat the stimulus, or in other words, to reduce the patient's forces, judged to be in excess.

Definition of Tolerance.

Tolerance only Apparent.—While in the healthy individual 5 to 10 centigramme doses of tartar emetic (1 to 2 grains) determine evacuations by the stomach and often by the bowels, in the sick man, especially in the pneumonic patient, 60 centigrammes (10 grains) and even more may be taken without acting either on the stomach or intestines, and without troubling the functions of sensibility or of movement. This is what is called *tolerance*,—I add the word *apparent* or *phenomenal*, for it is simply a case of difference of action of the same medicine according to the dose, whether small or large, in which it is administered. It is but a difference of phenomenality, that is to say,—disappearance of the emetic effect before the general effect; the supposed tolerance is all a delusion.

Real Tolerance.—This is not the only medicine, nor is pneumonia the only disease of which tolerance may be predicated; it has long been known that opium may be given with impunity in ten times the ordinary doses in certain grave affections of the nervous system, such as in chorea (generalized and persistent), in tetanus, in cerebro-spinal meningitis, and in hysteria. It is well known, too, that digitalis has been given in immense doses (tablespoonful doses of the tincture), by certain English practitioners.

Tolerance an Essential Therapeutical Property.—But that which has thus far been a matter of physiological interest only, becomes a fact of extreme importance in the treatment of febrile diseases and in grave diseases in general. Here the necessity and utility of large doses is apparent. If you prescribe for a pneumonic patient the same dose of digitalis—two to three grains a day of the powder, for instance—which you would order for a cardiac patient, you will obtain no effect either on the heart or on the temperature. When you have before you a patient suffering from pneumonia, especially if the febrile dyspnœa be pronounced, if the nutrition has undergone a thorough impairment, you will need to give eight, ten and even twelve grains a day to obtain an antipyretic effect. It is the same with sulphate of quinine, which must be given in half drachm doses a day if you would see the least therapeutic action; this is the law of tolerance in grave conditions, especially in hyperthermia. Here we see

the operation of a physiological phenomenon in the pathological world; here we observe an instance of the peculiar reactions of the sick organism to remedies, which to be adapted to the morbid condition must be given in doses far exceeding those necessary to produce certain definite results in the normal physiological state.

Hypotheses respecting Tolerance in General.

Pathological Physiology.—How shall we conceive, how interpret this anomaly, which seems to contradict all the laws of experimental physiology? The first idea which presented itself to the mind of clinicians, was that remedies act differently on the sick man and on the well man, and that experimentation ought therefore to be relegated to the domain of clinical medicine; this is a mistake easily pointed out.

One may say, at the most, that the medicament has for the sick person, other effects and consequences than for the healthy person. There is no such thing as pathological physiology, but there is, as Claude Bernard has said, a physiology in pathological conditions; the disease has not created any new thing; the patient lives with the same organs, but their functionating has undergone, by the morbid process, certain modifications which may be called quantitive; these variations are always found in a rudimentary state in the healthy organism, but they undergo multiplication or diminution in the state of disease, and to such an extent, that certain pharmaco-dynamic effects are rendered nil, while others are exaggerated, as is easy to prove by the instance (before given) of pneumonia treated by tartar emetic or digitalis.[1]

Want of Absorption of Medicaments.—In order to extricate themselves from the difficulty, physiologists have imagined that the poison is not absorbed in and by the digestive organs, that the absorptive function of the lymphatic vessels too is annihilated, and that consequently the medicine does not enter the blood. It is easy to prove the contrary; in pneumonic patients treated by tartar emetic the metal is found in the urine as well as in other secretions during life, and after death in the tissues, which almost all degenerate fattily by reason of the action of the antimony, and it is the same with regard to digitalis. Absorption does not fail, except when the circulation itself fails, as happens in cholera,

[1] Dujardin Beaumetz (Leçons de Clinique Therapeutique, vol. iii., p. 10) explains this tolerance somewhat differently. Under the influence of certain modifications such as those determined by alcohol, the typhoid fever or pneumonic virus, patients acquire a certain immunity from the ordinary action in ordinary doses, of a great number of medicaments. We can, he says, give a physiological explanation based on a curious experiment of Claude Bernard and Paul Thénard, which consisted in subjecting some hares to the influence of ether, then in injecting into their cellular tissue a quantity of anhydrous prussic acid; as long as the animal remained plunged in the anæsthetic sleep one might administer without producing poisoning, considerable doses of prussic acid, but toxic symptoms manifested themselves immediately when the animal recovered from the ether. This experiment clearly shows that where a nervous element is influenced or modified by one medicinal agent it does not readily submit to the action of another medicament. It is the same in the case of the pneumonic patient. The nervous system is oppressed, it may be overwhelmed, by the virus of pneumonia; it has lost its original susceptibility to ordinary medicines, and to obtain certain therapeutical effects from quinine, digitalis, etc., these remedies must be given in massive doses. The tolerance is, therefore, simply diminished susceptibility, due to profound modifications of a paralytic nature, in the tissues, and especially in the nerve centres.—Trans.

where the dehydration of the blood prevents this liquid from circulating; at the commencement of this disease all medicines are rejected by the digestive passages, although morphia hypodermically injected still has its usual effects; but as soon as the disease has arrived at the period of cyanosis, that is to say, when the water of the blood has been evacuated by the dejections, when the blood has become, so to speak, stagnant and charged with carbonic acid (which accounts for the cyanosis), all absorption is arrested.

One may give immense quantities of opium, of strychnine, or of any medicine whatever, and the physiological effects are absolutely nil; it is no longer *tolerance*, for in this state medicines are no longer absorbed and therefore are inert. Hence, abstraction being made of this condition of the system peculiar to Asiatic cholera, where we do not see true tolerance exemplified, absorption is ordinarily well performed; the medicament enters the circulation, where it produces its maximum effects on the functions of nutrition, which, perhaps, it enhances, and its minimum results on certain nervous functions which, perhaps, it abolishes.

Failure of Elimination.—It is in order to explain these violent anomalies that certain theorists have imagined a failure in elimination. The glandular organs, they say, no longer secrete and eliminate the poison as in the natural state; it accumulates in the system and is largely responsible for any untoward issue. This cannot be true, for repeated analyses of the urine and other secreted products of the economy, in these conditions, have shown the presence in those fluids of the *tolerated* medicament.

It now remains to determine the conditions of tolerance, a subject which has not yet been much studied.

Conditions of Tolerance of Medicinal Substances in Pneumonia.

From what has just been said, it is evident that for the manifestation of this tolerance there must be a disease of more than ordinary severity, and we have the most striking example in grave pneumonias.

We do not witness tolerance in simple bronchitis, in influenza, in broncho-pneumonia as in infectious pneumonia; moreover pneumonic patients at the onset, or those who are not seriously affected almost never manifest it. Grisolle formally asserts that tolerance is never established the first day; tartrate of antimony then provokes vomiting and diarrhœa. The second day, but especially the third and fourth, or the fifth at the latest—it unequivocally declares itself, in at least half the patients; why these differences? It is clear that it is only after the second day that the disease is pronounced, or in other words, may be said to have taken possession of the organism, but here is the dominant fact and which has not thus far been well understood; in twelve cases Grisolle saw tolerance established from the very commencement and nine of these cases were fatal. Hanot makes this appropriate remark: "We see that tolerance at the onset is not a thing to be desired;" the reason of this, then, is, that an extraordinary gravity of the disease, without which there would be no immediate tolerance, is thus indicated. It is not the tartar emetic which produced this mortality of three out of four, but the toleration was simply an index of a situation so perilous that no kind of medication would have averted the disastrous result. One may then say that the graver the pneumonia the greater the necessity for large doses, which are then easily borne.

Pneumonia Variously Treated.—It is the same with other means of treatment; thus digitalis and alcohol are infinitely better tolerated by the pneumonic patient than by the healthy individual. The physiological effects are wanting when ordinary doses are given. The digitalis does not slow the pulse and the alcohol does not produce stimulation, except in considerable doses, which alone are capable of causing therapeutical effects.

Interpretation of Tolerance in Pneumonia.

What takes place, then, in the organism of the pneumonic patient when the morbid process has arrived at its maximum? We have a raging fever, cardio-vascular depression, threatening asphyxia, and eventually prostration of all the vital forces. The hyperpyrexia produces in the ganglionic nervous system profound perturbations which find expression in the disordered innervation of the heart and blood-vessels.

The hyperthermia also menaces the heart with fatty degeneration; it has, moreover, a detrimental action on the nutrition and innervation of the principal organs, and this is why they no longer react normally and regularly under the influence of pharmaco-dynamic means.

The asphyxia which results from the extension of the pulmonary inflammation exercises an influence still more marked on the economy, for the blood impregnated with carbonic acid has a paralyzing effect on the functions of the peripheral and central nervous system; the excito-motricity of the spinal cord cannot manifest itself except in the presence of oxygen, and it is annihilated by carbonic acid; Sctschenow has proved this. There results, on the part of the stomach, a depression of reflex power which opposes emesis:—as for the patient, there is insensibility to the action of medicines proportioned to the degree of the asphyxia, or in other words to the intensity of the morbid process. In fine, the prostration goes on augmenting; the muscular system of organic life, the myocardium, the unstriped muscles, suffer deterioration, and digitalis and antimony only enhance the organic dilapidation. If you give these medicines in small doses they are without effect; if you prescribe large doses you only hasten the ruin of your patient. The physician is thus put in an unfortunate dilemma—the more unfortunate the more faith he has in the antithermic or contra-stimulant treatment. Little doses are powerless, and maximum doses, sufficient to arouse the prostrate and languishing organism, are dangerous, in that they add to the burdens under which the economy is already struggling. The patient smitten by the disease and by the remedy, seems to have a poor chance in the seemingly unequal contest. If, however, his powers of recuperation are sufficient to carry him through in safety, the organs and tissues by degrees reassert their rights, the pneumonia begins to undergo resolution; now, singularly enough, we find our contra-stimulant medicine no longer tolerated; a proof that the organism, when under the inhibiting pressure of the malady, did not respond to the toxic doses because not in a condition to respond to them.

In the case of quinine and alcohol, as we shall see farther on, similar phenomena are witnessed.

Conditions of Tolerance in other Diseases.

In the other febrile diseases it is still the hyperthermia which compels the physician to give massive doses of the medicament on which he relies.

The same want of action of remedies is seen, as before said, in certain grave diseases of the nervous system, such as tetanus and chorea. The condition of pathological maximum always necessitates an unwonted employment of pharmacodynamic resources. Other causes lend their influence in these difficult cases to lessen reaction and make ordinary medicinal doses of little avail.

CHAPTER XXV.

STUDY OF ANTIPYRETIC MEDICAMENTS AND MEANS.

Digitalis.

History. Preparation. Doses. Intolerance.—Guided by his experimental researches, Traube was the first to apply digitalis to the treatment of pneumonia. He was followed by Hirtz and his pupils, by Saucerotte, Gallard, then by Jaccoud, who clearly laid down the indications of this treatment. In Germany the number of practitioners who advocate the employment of digitalis in pneumonia is ever becoming less and less, by reason of the inferiority of this drug to other antithermic medicaments. The only preparation which has been everywhere in usage is the infusion of the leaves; neither the alkaloid nor the glycerides ought to be thought of in this connection, as they are uncertain preparations and are not to be relied on for an antithermic effect. It is nevertheless to be remarked that the infusion contains two of the principal glycosides, digitaline and digitaleine, which are soluble; it does not, however, contain the digitoxine, which is the most active part. We know the dose to prescribe, but we cannot tell if this dose, which in pneumonia is necessarily large, may not provoke symptoms of intolerance, such as nausea and vomiting. It is certain that in quantities of twenty grains toxic phenomena may be produced, such as coldness of the extremities and collapse.

Physiological Effects.

Slow and Cumulative Action.—Certain grave inconveniences are incidental to the usage of digitalis; I refer especially to its slowness of action and its cumulative tendency. Schmiedeberg attributes the first to the difficulty of absorption of the glycerides by reason of their slight solubility. As for the accumulation, he considers it owing to want of elimination by the kidneys, disturbed in their functions as a consequence of excessive use of the medicament. It is always the case that it does not manifest its maximum effects in pneumonia till at the end of twenty-four or thirty-six hours, and this circumstance suffices to restrict its employment; if you prescribe it the third or fourth day, the curative effect, supposing that any such effect is realized, will be confounded the following day with the natural crisis. On the other hand, if you continue the digitalis for several days, it will produce more marked effects at the end of this time than at the beginning; you should, therefore, be armed with patience, and await the definite result without continually augmenting the dose, as you would thus intensify the evils of accumulation, which are often noticed for several days after cessation of the treatment.

Action on the Heart.—It is important to know exactly the action of digitalis on the heart. Ought it to be reckoned among the poisons or

the tonics of the heart? The labors of Vulpian, Dybkowski and Pelikan, Böhm, Schmiedeberg and Williams have taught us that in the heart of the frog there is first observed augmentation of the volume, that is to say, of the intensity of the contractions, with prolongation of the diastolic phase. Then, after certain partial irregularities of action of the different portions of the heart, one part of one ventricle after another is seen to lose the power of entering in diastole, so that finally all the ventricle is in a state of systolic contraction; the muscular substance is then affected. Excitation of the vagus does not bring back the heart to diastole, but the pressure of any liquid whatever introduced into the ventricle may restore the contraction momentarily; then the organ dies. In warm-blooded animals phenomena of the same kind are observed, but the heart is not arrested in systole; the alteration of the muscle does not go as far as this.

Action on the Energy of Pressure.—The energy of the heart manifests itself by the intra-vascular pressure, which is augmented. This may be recognized in man by the aid of the sphygmograph; in animals by the manometre.

It has even been maintained in France that this excess of tension in the blood-vessels after digitalis is primary, and that it reacts in its turn on the heart to augment the contractile force of this organ. Klug, in Germany, has gone even farther. He believes that there is both an excitation of the vaso-motor centre causing constriction of the blood-vessels and an alteration of the muscular elements of the vascular walls. But we have only to suppress the blood-vessels in order to note the independence of the exaggerated contractions of the heart. (Böhm, Williams.) The direct effect of digitalis on the muscles of the blood-vessels being moreover impossible to demonstrate, one may be warranted in concluding that the blood pressure augments only in the ratio of the action of the heart.

Action of Digitalis on the Heart's Rhythm.—At the same time that the heart beats with more force, it beats with less frequency. It is just this physiological effect, namely, this slowing with increase of contractile force which is utilized in the treatment of pneumonic fever.

Action of Digitalis in Toxic Dose.—But do not forget that if you attain the toxic dose, it is quite different; the pulse is enormously accelerated, and the blood pressure ends by falling.

Mechanism of the Divers Effects of Digitalis on the Heart.—To explain these successive effects, Traube concluded that we must attribute these phenomena to the influence of the pneumogastric nerve, whose excitation by the drug slows the heart, but whose paralysis by toxic doses accelerates the pulsations. One may, however, with impunity section the trunk of the vagus, and the same physiological effects will follow moderate or toxic doses. It is in the heart itself that we must look for the origin of this succession of opposite phenomena; there is in the substance of that organ a depressor ganglion which acts like the pneumogastric nerve. This is so true that, if in the frog, you paralyze this ganglion by atropine, the effects of digitalis are annihilated, and there is no longer either slowing or acceleration of the beatings. In the last period of the digitalis intoxication, the heart muscle is itself affected in its intimate texture; it is then that the heart is arrested in a state bordering on tetanic contracture. As for the pressure, it follows in its different phases the excitation or paralysis of the intra-cardiac depressor centre, augmenting with the one and diminishing with the other.

This fact, then, remains indisputable, that digitalis in moderate doses is

not a poison of the heart, but that, on the contrary, it fortifies its contractile power, and energetically maintains the intra-vascular tension.

Action on the Temperature, the Oxidations, the Urine.—The action of digitalis on the temperature is not as pronounced as the effect on the heart. By reason of the increase of arterial pressure, the circulation is energized at the periphery, but for this very reason heat is more promptly and more readily radiated away, whence results a certain degree of loss of the internal heat. (Akermann.) The oxidations are rendered more active as long as the pressure remains high; urea is then eliminated in greater quantity, diuresis is established, and persists till the pressure is lessened.

Digestive Troubles.—All the series of phenomena, cardiac, vascular and thermic, may undergo evolution without digestion being disturbed, but it is not always so, for nausea and vomiting are often observed even after moderate doses, and this puts a check on the further administration of the digitalis.

Therapeutic Effects.—The physiological effects are seen in even a more marked degree in pathological states. As a result of doses of from 10 to 13 grains, the pulse becomes slowed, the arterial pressure is increased; these phenomena, however, do not appear till at the end of from twelve to twenty-four hours. The temperature does not fall till after from thirty-six to sixty hours (Bernheim); the respiration also then undergoes favorable modifications, and the febrile dyspnœa tends to disappear. From this moment the medication may be discontinued, for the effects obtained last often for one or two days after the defervescence. It is the more important to cease the treatment at an early date, as tolerance is often established only with difficulty, and is interrupted by severe digestive troubles, among which are obstinate vomitings which nothing can control.

If you persevere, and, for a stronger reason, if you exceed the dose of fifteen grains, all the advantages which you have gained are lost, and the opposite condition is now realized; the heart gives way, and runs riot in its pulsations; the blood pressure falls, and often the patient lapses into a marked state of collapse with coldness of the whole body.

Therapeutic Results.

Thomas' comparative statistics[1] show us that the mean duration of the disease when treated by digitalis is the same as when treated by expectancy; digitalis cannot prevent the fatal termination. If, however, death threaten to take place by the hyperthermia, or by the extreme frequency of the pulse, without the danger being immediate, so that one has time to wait the always slow effect of the medication, the indications are clear and precise; no other medicine can fulfil them better; it is the cardiac tonic par excellence; is also a refrigerant, but more uncertain than quinine and antipyrine, of which we shall soon have occasion to speak.

Nevertheless digitalis is of great service in infantile therapeutics, according to Duclos and Bergeron. The daily doses of from one to two grains of the leaves for a child of from three to six years of age, and double the quantity for older children, contribute to maintain the cardiac power.

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[1] Arch. f. Heilkunde, 1875.

Quinine.

General Considerations. Doses.—Sulphate of quinine is a powerful antithermic and is at the same time a precious cardio-vascular medicament. It is, in the second place, a parasiticide which nothing can replace in the treatment of malarial affections. It is lastly a poison to certain histological elements, among others the white corpuscles, and on this account has been (strangely enough) considered an antiphlogistic. (Lépine.) Its antithermic and cardiac properties have alone been utilized thus far in the infectious pneumonias, for which we as yet know of no specific remedy. Its anti-parasitic power in these pneumonias is under dispute, but is incontestable in the true malarial pneumonias.

Doses.—Sulphate of quinine is of no effect in pneumonic fever except in large doses; in this respect it resembles tartar emetic and digitalis. Vogt prescribed it in half-drachm doses; Liebermeister gives from twenty to thirty grains in the space of an hour to bring down the temperature to 100° F. and even below. Jurgensen has given as much as seventy-five grains at one time; such a dose is plainly dangerous, for the point of extreme tolerance is reached when somewhat less than a drachm has been given, as we have often noted; beyond that quantity mortal collapse may set in. Below twenty-three grains the refrigerant effects are nil.

Mode of Administration.—To obtain any remedial action you must give successive doses which must be near together. If you give the necessary quantity in fractional doses during the twenty-four hours, the medicine is eliminated by the urine before there is at any one time enough of it in the blood to produce the desired effect. There are physicians who are afraid to exceed one gramme (fifteen grains) in the twenty-four hours. They obtain no result and complain of the remedy as inefficacious, whereas if they had given two grammes (or thirty grains) they would have seen a marked effect. These thirty grains should be administered in two doses, and not in fractional doses, for breaking up this quantity into ten parts is equivalent to a complete nullification of its physiological effects. You may prescribe the first dose of fifteen grains early in the morning, and the second late in the evening,—after nine o'clock—this is my favorite method; or you may give, as Liebermeister recommends, the first dose of one gramme at three o'clock P.M., and the second dose two hours later.

The same general directions apply to feeble persons, as well as to those that are vigorous. There is no way by which we may judge beforehand of the influence of the individual force or constitution on the action of the remedy. Age alone creates a certain difference. The child between the ages of two years and five (before two years lobar pneumonia is rare) bears well doses of five and even eight grains; to a child of from six to ten years of age one may without fear prescribe fifteen grains as a daily dose. The difficulty consists in the mode of administration; while in the case of an adult fifteen grains in powder or in capsule may be supported, in the child it is often necessary to have recourse to the quinine lavement, which is generally absorbed by the intestinal mucous membrane.

The doses indicated ought to produce a refrigerant effect at the end of from five to seven hours at the latest. If the temperature does not fall, it is because you have to deal with a case of the most extreme gravity which nothing can cure. Bear in mind that the physiological effect of medicaments may be nil by reason of the deficient action of the organs, and the moment you test their vitality they do not respond; we then witness abso-

lute intolerance instead of that tolerance of large doses, so characteristic of the grave specific phlegmasias.

Antipyretic Properties—Refrigerant Action.

Refrigerant Action.—Large but non-toxic doses produce, even in the healthy person, a quite marked fall of the temperature, which ceases to present its usual diurnal variations. (Kerner.) This refrigerant property is especially noticeable in fevers, but after a different fashion; if you produce in animals an experimental fever, you determine, but only after considerable doses of quinine (Manassein), a marked diminution of the febrile heat, and at the same time an improvement in the general condition. (Binz, Naunyn, Quincke.) In the pneumonic patient this effect is no less marked; one succeeds by the massive doses indicated in reducing the heat a degree and a half, but at the end of from eighteen to twenty hours it tends to return to the previous thermometric height.

Mechanism of the Refrigeration.—The phenomenon of refrigeration is easily explained. It is due, not to excessive radiation and loss of heat, but to a diminution of heat-production (*i.e.*, a lessening of calorifiant power). In an animal poisoned by quinine, whose spinal cord has been sectioned, subsequent heat generation, such as is wont to take place even for a time after death, is prevented. The refrigeration, then, depends neither on innervation nor on the circulation; it can only be attributed to a lessening of the chemical oxidizing processes of the economy by the quinine. The proofs of this are furnished us by observations of phenomena pertaining both to health and disease.

Diminution of the Oxidations.—Ranke, Kerner and Prior have found that in a healthy individual urea diminishes from 17 to 24 per cent., uric acid 8.2 per cent., the chlorides 8.3, and the phosphates 13 per cent. under the influence of quinine.

The proportion of carbonic acid eliminated falls also 9 per cent., and that of oxygen absorbed undergoes a reduction in the ratio of that of the carbonic acid exhaled.[1]

Cardio-Vascular Properties—The cardio-vascular properties reveal themselves in animals under two phases. When fifteen grains of quinine are introduced into the stomach of a dog, you observe at first an acceleration of the pulse with temporary irregularities and considerable augmentation of the pressure; then this pressure, which had risen to twenty-five centimetres, falls to its normal degree and even below. (Sée and Bochefontaine.) The heart presents at first a functional excitation which manifests itself by an increase of the systolic impulse, coinciding with a sensible diminution of the frequence of pulsations; finally the activity of the heart is lessened in its turn, unless a new dose of quinine is administered. In the case of the fever patient two phenomena are observed of the greatest importance—

1. The energy of the heart is preserved intact, augments even, as is indicated by the abrupt and long line of ascent of the sphygmographic tracing. This is what we have observed in all the cases.

2. The dicrotism of the pulse is suppressed; this we have observed in the clearest manner in typhoid fever, where the dicrotism is so pronounced by reason of the hyperthermia. It is well known that dicrotism is the

[1] Strasburg.

index of a diminution of the blood pressure; it marks itself upon the line of descent of the sphygmographic curve, by an undulation which is only the lifting of the artery by the retropulsive blood wave. Now, everything which diminishes the vascular tension, as does fever heat, augments this undulation, that it is to say the dicrotism; if, then, we succeed in suppressing the fever heat by the quinine the dicrotism disappears.

Hence everything concurs in demonstrating that sulphate of quinine is a tonic of the heart, whose contractile energy it augments while it prevents or stays the relaxation of the blood-vessels which is the cause of the dicrotism. No other medicament possesses this double property, and it is apparent from the following parallelism how and why sulphate of quinine opposes the fever.

Antagonism between Quinine and Fever.

The febricitant always presents the following characteristics:	The febricitant, under the influence of quinine, presents certain phenomena as follows:
1. The combustions are exaggerated. The exhaled carbonic acid augments sensibly.	The combustions are manifestly diminished. The formation and elimination of carbonic acid prove it.
2. The urea, which represents the product of combustion of the tissues and albuminoid matters, is often largely augmented.	The urea diminishes 15 to 24 per cent.
3. The pulse is accelerated.	The pulse is slowed.
4. It is also constantly dicrotous.	The dicrotism almost always disappears.
5. The temperature is from 100° F. to 105° F.	The temperature falls a degree and a half.
6. The heart is inclined to become feeble.	The heart acquires stronger contractile force.

Therapeutic Effects. Comparison with Digitalis.—Quinine, which continues to be employed in Germany, and of which Lépine extols the effects in the treatment of pneumonia, constitutes an antithermic which presents incontestable advantages over all other means of this kind, with the exception perhaps of antipyrine, to the refrigerant action of which, being immediate and at the same time perfectly safe, we shall have to call attention. It is not, like digitalis, slow, ordinarily too tardy in its effects; it has not the inconvenience of being cumulative in the organism; its refrigerant power is more marked than that of digitalis; moreover it not only sustains the contractile force of the heart and blood-vessels, but it is also a sparing medicament (*medicament d'épargne*), which prevents to a certain extent the oxidations of the economy, and thus sustains the general forces. The duration of its action, it is true, is less than that of digitalis, but one can remedy this inconvenience by continuing the doses indicated. The comparison with cold baths is still more to the advantage of quinine; the proof will readily appear.

There remains veratrine, which is in reality a poison of the heart, the minute study of which will show the danger of resorting to it in doses such as will produce any appreciable result. If, then, antipyretics ought to be employed to restrain the hyperthermia and its grave consequences, it is to quinine and to antipyrine that you should have recourse.

Antipyrine.

History, Chemical Properties, Doses.—Among the products of the quinoleine series is reckoned, besides kairine, which presents too many inconveniences for practical use, antipyrine, discovered by Knorr and experimented with by Filehne. It is a white substance, easily soluble in water, of a slightly bitterish and aromatic taste. It is obtained by synthesis from aniline and from acetic ether, and is a derivative of quinoleine by oxidation of pyridine.

Filehne attributed to it very energetic antipyretic properties, which have been verified by Guttmann, Alexander, May, Falkenheim and Rank.[1]

Doses and Modes of Administration.—Ordinarily antipyrine is administered in solution in water or in wine. You dissolve 10 parts of this substance in 150 parts of water, and regulate the doses in such a way that the patient shall take thirty grains, then forty-five grains of antipyrine within a brief space of time; the whole quantity to produce an antipyretic effect being about seventy-five grains.

It has also been prescribed in hypodermic injections; and is so employed by Rank; one gramme (fifteen grains) being dissolved in about three times its weight of warm water and injected subcutaneously.

Effects in Febrile Diseases.—Filehne has succeeded, by means of the mode of administration indicated above, in reducing the temperature in febrile affections, even when it was very high, to the normal figure of 98½° F.; generally the diminution is from half a degree to two degrees. The effect is produced gradually, with or without sweats, and attains its maximum at the end of from 3 to 5 hours, persists from 7 to 9 hours and even sometimes 20 hours; then the temperature mounts up again without the accompanying chill, such as takes place in the case of kairine. Guttmann, who has employed it in 27 cases of fever, has confirmed these results, which have been verified by all the clinicians.

When the fall of the temperature is very marked, there is often produced considerable SWEATING, sometimes even excessive perspiration precedes the fall of the fever.

The PULSE comes down at the same time as the temperature; sometimes there is not absolute parallelism, and often the fall of the pulse is not noticed till later; this confirms the experiments of Fick, who has seen the hypothermia act on the respiratory centres while the vaso-motor centres and the centres of cardiac innervation remained unaffected.

In a few cases, VOMITING at the onset has been observed, but generally the medicine is well supported.

No trouble is produced in the functions of the URINARY organs.

Duration of the Apyrexia.—The apyrexia continues for fifteen hours; this duration is less in pneumonia than in typhoid fever.

In INTERMITTENT FEVER the attack may be cut short when the medicament is administered at the onset; but the paroxysms reappear, and antipyrine can never take the place of quinine in this disease.

Personal Observations.—The small quantity of antipyrine which I have been able to procure has enabled me to witness astonishing results, especially in the fever of tuberculosis; less marked, however, in typhoid

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[1] Vide Fortschritte (Friedländer), No. 14, 1884. See also Miller in Centralblatt, Aug. 16, 1884.

fever and pneumonia. Being more easy to administer, more prompt in its action than quinine, and without any inconvenience, it appears certainly to claim superiority over the latter.

In fact all observers are unanimous in declaring that antipyrine constitutes the most faultless, the most certain of antipyretics. Does this kind of treatment, even though one succeeds all through the disease in keeping the patient cooled down to the normal, suffice to *cure* the patient or, at least, avert the dangers? Here is summed up the entire question of hyperthermia.

Cold Baths.

History. Mode of Administration.—It was Vogel of Berne who first had the thought and the courage to apply cold baths to the treatment of pneumonia. Liebermeister was not slow in following his example, and he pretended that by this medication the mortality was reduced from 24.4 to 8.8 per cent. Mayer and Fismer announced the same results, but it is Jurgensen especially, who is the great advocate of this method, by which he did not fear to treat his little daughter, aged 19 months.

The mode of administration is almost always the same; as soon as the axillary temperature of the pneumonic patient exceeds 103½° F. he is plunged into a bath of 22° to 24° Réaumur (81° to 86° Fahrenheit), which is cooled down to 16°, (68° F.).

The temperature of the patient falls at the end of ten minutes, but two hours afterwards the thermometer mounts up again to the previous level; it is then necessary to resume the treatment and often as many as a dozen baths a day have been given by partisans of this mode of treatment. Others, more moderate, limit themselves to four a day, and keep the temperature of the water at 20° Réaumur (87° F.), which is but little below the tepid heat.

Physiological Effects. Therapeutical Results.—Cold baths lower the temperature only temporarily, but beneficial effects have been attributed to them; according to Strumpell they facilitate the respiration, which becomes slower and more profound; they promote expectoration, and improve the general condition, procuring for the patient a sensation of *bien être* and greater comfort; it is, then, to calm the dyspnœa and nervous disturbance that they are recommended. But this slight benefit is bought at a dear price. All the advocates of this system of treatment, among others, Fismer and Jurgensen, have pointed out the danger of *accelerating* the respiratory movements, of provoking cyanosis, and even syncope, and they therefore recommend to the patient to take before the bath two or three spoonfuls of strong wine (Jurgensen); this is evidently the most rational part of the treatment.

Indications, and Contra-indications.—The final result has been that the cold bath is no longer prescribed as a part of the regular routine treatment of pneumonia; it is reserved now for the ataxic or adynamic form of the disease, and is interdicted in the case of alcoholics, in old men and all the pneumonias which are very extensive. Simple pneumonia in the adult can certainly do very well without such heroic treatment.

Comparison with Quinine.—The comparison with sulphate of quinine is decidedly in favor of the latter. The cold bath is at the best a transient though speedy refrigerant, which augments all the com-

bustions of the economy, while quinine is a true antipyretic which retards the oxidations and thus indirectly spares the forces.

The cold bath has no effect on the circulation, unless it be at times a depression of the contractile force of the heart, and even collapsus. Quinine is a heart tonic, which markedly raises at a given moment the blood pressure so as to cause the dicrotism to disappear, when it exists, as in the grave pneumonias.

Veratrum and Veratrine.

History. Chemical Properties. Doses.—Veratrum begins the series of musculo-cardiac medicaments, or rather poisons, for all the preparations of the veratrums and their alkaloids present a great similarity. Their action is energetically and uniformly manifested on the muscular system, the cardiac muscle included, which are directly affected without the intervention of the nervous system. It is by reason of its effects on the heart that veratrine has been classed among the medicaments which are cardiac and at the same time antithermic; it does not, however, cause any lowering of the temperature, and what effect it has on the heart is not that of energizing the action of the later, but of depressing or paralyzing it.

Preparations.—In America physicians have long employed the *veratrum viride*, which has been considered almost a specific in febrile maladies, especially pneumonia. It is prescribed in the form of tincture, in the dose of 4 to 20 drops; collapse is likely to follow the larger dose. In chronic diseases certain authorities recommend the tincture in gradually increasing doses—beginning with one and not exceeding 20 drops; it is safe enough when administered in this way, provided its administration be stopped when nausea supervenes. It is in this manner that I have prescribed it in Basedow's disease, just as Skoda and Græfe were in the habit of giving it, and with success in this affection, called exopthalmic goître, and which is in reality nothing but a tachycarditis [1] by paralysis of the pneumogastric. Conjoined with a judicious use of hydrotherapy, this medication is preferable to digitalis, which in the end accelerates the heart's action. If in suitable cases I have of late discarded veratrum, it has been to substitute for it a more efficacious medicament, namely, convallamarine.

Veratrum Album. Veratrine.—All the veratrums contain several active principles, among others, veratrine; jervin and veratroidin have also been mentioned, but these have not been much studied; it is veratrine which especially predominates in the veratrums, and is also met with in sebadilla, in colchicum, and in veratrum lobelianum.

Doses.—Veratrine alone is used at the present day. Although soluble in alcohol, it is almost always given in the form of pills or granules each containing a milligramme ($\frac{1}{60}$ grain) of veratrine. It is prescribed in gradually increasing doses till from 10 to 12 milligrammes a day are taken; these doses are, however, often attended with vomiting, troublesome diarrhœas, sometimes prostration, or even a collapse, which is so frequent and readily foreseen, that the partisans of the veratrine treatment find themselves obliged to associate this medicament with wine or alcohol to sustain the forces of the patient. (Kocher, Bernheim.)

Physiological Effects. Action on the Striped Muscles.—The

[1] A term coined by Prof. Sée to indicate a condition of the heart due to paralysis of the vagus, and indicative of one of the prominent phenomena of the disease, *i.e.*, exaggerated activity of the pulsations.—Tr.

most remarkable and the most constant effect of veratrine is its action on the heart and other striated muscles, which manifest an extraordinary retardation of the curve of contraction; this is clearly apparent from the researches of Kolliker, Bezold and Hirt, of Prévot of Geneva, of Marey, etc.

If you examine, in the frog, and especially in the *rana temporaria* (Prévot), a muscle under the influence of veratrine, you observe that the contraction takes place as quickly as in the normal state, but the muscle does not relax, or resume its normal length except very slowly, so that the entire duration of the contraction is five or six seconds. From this it results that the voluntary movements of the animal are much retarded and as it were forced, so that some have even believed in the existence of a species of tetanus; this is, however, a mistake, for there is no such condition, tetanus being dependent on an excitation of the spinal marrow. Now all this series of phenomena, which begins by a sort of contracture, and ends by resolution with loss of muscular excitability, is absolutely independent of the nervous system, central or peripheral. You may cut the spinal cord without modifying this singular manifestation; you may section the nerves, you may paralyze with wourara the motor terminal expansions, without modifying in any degree the production of these strange contractions. In fact the muscles alone are affected, and they are affected directly; what proves this is that the contraction is accompanied by a more marked production of heat than in the normal state, and consequently by a greater activity of nutritive exchanges (Fick and Böhm); the muscle under veratrine is so altered by the poison that it loses to some extent its elasticity. (Rossbach and Anrep.)

In the higher animals poisoned by veratrine the muscles also become the seat of that same prolonged though not tetanic rigidity, and later manifest extreme feebleness of contractility.

Depression of the Heart Muscle.—The cardiac muscle undergoes exactly the same alteration as the rest of the transversely striated muscular system. The heart of the frog taken from the body, and put in communication with a suitable enregistering apparatus, exhibits often a marked slowing of the pulsations, systolic contractions of greater and still greater length, and finally pauses of from twenty to thirty seconds, so that the number of the pulsations is reduced one half before the organ finally ceases beating, which is not till after several hours; the heart becomes inexcitable under the most energetic irritants; electrical stimulation of the vagi nerves, of the venous sinuses, is equally without effect; the special action of muscarine, which arrests the heart by excitation of the pneumogastric, in no way affects the heart which is veratrinized; eserine, which excites the same nerves, atropine which paralyzes them, curara, which acts on the intra-cardiac ganglia, do not modify in the least degree the type of contractions caused by veratrine.

In the hare and the dog one-twelfth to one-sixth of a grain of veratrine injected under the skin determines immediately a slowing of the cardiac beatings, and at the same time a considerable diminution of the blood pressure, finally irregular pulsations and paralysis of the heart. This is not the result of excitation of the terminal filaments of the vagi, as Bezold and Böhm thought. We know already by the experiments on the veratrinized heart of the frog that it is a direct effect of the poison on the myocardium.

Respiratory Troubles.—The respiration undergoes only transitory

modifications. At first there is acceleration, which rapidly yields; under large doses, there is at once retardation, and finally a compete arrest of respiration by reason of paralysis of the respiratory centre and of the pulmonary pneumogastric. The type of the respirations is also modified; it becomes, as it were, convulsive, with long expiratory pauses, which resemble those of respiration after section of the vagi. All these phenomena have no special interest, and are less conspicuous than the following.

Digestive Troubles.—Veratrine acts almost always injuriously on the digestive organs. It produces a sensation of heat and formication, then nausea, violent vomiting, and diarrhœa with colic and tenesmus, while the autopsy does not reveal the least inflammatory lesion—which proves that these phenomena depend on excitation of the nerve endings in the stomach and intestine.

To sum up: all the action of veratrine is concentrated on the muscular system and the heart; the digestive troubles are accidental or secondary.

Therapeutic Effects.—It is under the name of antipyretic that veratrine has been administered in all the fevers, and in pneumonia especially. Vogt and Kocher at Berne, Drasche at Vienna, Aran in France, brought this medication into vogue long before we had ascertained the physiological effects; the most elementary notion of which would have sufficed to condemn this cardiac poison. See in fact what is observed:

A notable fall in the beatings of the HEART. A corresponding retardation of the pulsations of the artery, lasting eight or ten hours; a very marked diminution of the intra-vascular pressure and sometimes of the respiration.

All the above takes place without any notable or lasting modification of the FEBRILE HEAT; in not half the cases is any refrigerant effect observed. In some cases a trifling fall of temperature is noted, which, however, does not last beyond six hours. (Kocher.)

Therapeutic Results in Pneumonia. Influence on the Local States.—The progress of the lesion may be arrested it is said, during the first days—this is simply the history of abortive pneumonias; when the lesion goes on to hepatization it continues to progress even when the pulse and temperature fall, there is no remedial action whatever. The mortality is in no way lessened under veratrine. As for the indications for its administration they are the same in all these debilitant medications. We have the usual hackneyed directions; we are told to give the veratrum to young and healthy individuals when the disease is little advanced, and when the fever is intense, but these are just the cases that get well by expectancy.

CHAPTER XXVI.

DYNAMIC MEDICATION.

This medication is composed of all the means destined to support the forces of patients under pneumonia. Besides ordinary foods, the most of which are inadmissible on account of digestive imperfection and failure, there is a substance which constitutes what may perhaps be called an aliment, certainly a sparing agency, and no less surely a means of sustaining the forces. I refer to ALCOHOL.

Clinical History. Doses.—After the publication of the work of Todd in 1860 the utility of alcohol in the treatment of fevers became in England, and soon afterwards in France and America, a veritable article of therapeutic faith. Lionel Beale, Broadbent, Drysdale, Anstie, Behier, Flint, succeeded in systematizing this method of treatment, which is certainly one of the most useful from all points of view.

In pneumonia the dose, as in the case of all the active medicaments, ought to be large to be efficacious. The famous potion of Todd is reduced in France to 60 or 80 grammes (℥ ij to ℥ iij) of brandy per day; it is not in this way, however, that Todd prescribed alcohol. He was in the habit of ordering half an ounce (15 grammes) of brandy every hour, which would give a total of 12 ounces, or somewhat less than a pint, a day; sometimes, however, the amount was increased to a pint, but the spirit was always administered in small quantities and often. This is, in fact, a pressing indication, for absorption being easy and the alcohol being eliminated as such, it is important to act in a continuous manner on the forces, and to impregnate, as it were, the nervous system which directs them. The administration of the liquor with water or milk is of less consequence than the ingestion of the total quantity and its regular distribution in fractional doses.

Nutritive Properties of Alcohol.—How does alcohol act? Is it an aliment which undergoes combustion in the economy, is it a sparing agency which retards denutrition without being itself destroyed? Alcohol is considered by certain authorities as a respiratory food, *i.e.*, like the hydrocarbonaceous aliments which are burned in the system for the production of heat. This is the opinion of Liebig and Bouchardat, and has been supported of late by the experiments of Zuntz and his pupils. If it is in fact proved that alcohol in substance is removed only in small quantity by the secreting emunctories, it would seem an irresistible inference that it is burned in the economy.

Now the intermediate products of its combustion, such as aldehyde and acetic acid, have not been detected, and it is the same with the carbonic acid of expiration, which is the final product of the combustion of alcohol; far from augmenting, as we should expect would result if alcohol

be consumed in the organism, there is real diminution of this gas in the expired air. All experimenters, from Perrin, Lallemand and Duroy to Wolfers, Simonosky and Schoumoff, are agreed on this point, which may be now regarded as settled. But although the products of combustion are not found, it is none the less certain that the quantity of undecomposed alcohol which passes out in the excreted liquids is very insignificant. It is unchanged in the blood (Perrin), it is eliminated as alcohol in the urine and by the lungs; but the more precise researches of Bodländer have shown that in man only about 3 per cent. is removed by the emunctories, to wit, 1.17 per cent. by the kidneys, 0.14 by the skin, and 1.89 by the lungs: all the rest, viz., 97 per cent., remains in the organism. A certain quantity fixes itself in certain organs—the liver, the muscles, the lungs, and especially the encephalon. (Perrin.) If, however, we reckon out this quantity, we shall not materially modify the total figure of 97 per cent. What becomes of the great bulk of the alcohol ingested?

One might be tempted to believe that if alcohol is not itself consumed, it may promote consumption of the tissues, and thus augment the organic oxidations, but this again is contrary to what takes place.

Alcohol Retards the Oxidations, as is proved by the diminution of urea in the urine. Zuler and Stabing insist on this point, and Riess has noticed a decrease of urea amounting to at least 22 grammes per day in febricitants. In this respect alcohol resembles fat, which also exercises a real economizing action on the albuminates, restraining their waste (Fokker.) By the ingestion of alcohol as well as of fats, the urea diminishes, and as a consequence, the weight of the body increases. It is only when the dose is considerable, that the urea remains stationary (Monk) or is even found to be augmented. (Wolfers.) Hence it may be said that alcohol, like fat, is a waste-restraining means. This is the verification of Voit's doctrine respecting *tissue saving.*

Alcohol Diminishes the Quantity of Atomistic Oxygen.—One of the most evident confirmations of this power possessed by alcohol of modifying denutrition is that the products of oxidation of ingested benzol, such as phenol, which is eliminated by the urine, diminish sensibly when alcohol is caused to be absorbed at the same time as the benzol. A hare to which is administered one gramme of benzol, instead of passing 0.248 of phenol, eliminates no more than 0.076 of this substance when alcohol is given to it at the same time. Hence alcohol diminishes considerably the quantity of atomistic oxygen disposable for the combustion of the albuminates;[1] it reduces disassimilation to the minimum; it is a medicament which is conservative of the integrity of the tissues. I had reason to say at the commencement that alcohol is perhaps a true food; certainly it may be regarded as an indirect aliment. If this be so, certain consequences follow; by retarding waste it sustains the forces.

[1] Nenky, Simonosky, and Schoumoff. According to the researches of these experimenters, under the influence of alcohol the quantity of substances combustible by oxygen diminishes from 50 to 75 per cent; fat and albumen undergo oxidation but imperfectly. Benzol oxidizes in the economy, giving rise to phenol, pyrocatechine, hydrochinon, etc. Outside the organism, these oxidations take place only under the influence of atomistic oxygen. In the organism, the products of oxidation of benzol give the measure of atomistic oxygen formed in the tissues. The decreased genesis of these products in animals after the ingestion of alcohol along with the benzol is a very significant fact. (See the Author's. Phtisie Bacillaire, page 501.)—Tr.

Dynamic Nervous Properties.—It is empirical observation which from all time has given conviction that alcoholic stimulants maintain and augment the powers of the economy, and on this conviction Todd's method is based. This great clinician made no pretension to cure pneumonia, but he aimed to furnish to the pneumonic patient the force necessary to struggle against the morbid principle and thus favor the conditions of recovery.

It is the nervous system which, by impregnation with the alcohol, is benefited the most in the struggle against denutrition; the oxidations of the nervous substance under the influence of alcohol are lessened, and this makes itself apparent in the diminution of phosphates in the urine. (Stabing.) Hence the veritable mode of action of alcohol consists in retarding destructive metamorphosis of the tissues, in maintaining the organs in a state of nutritive equilibrium, and in thus staying the wasteful ravages of fever and keeping the febricitant in possession of his muscular and nervous forces.

Antithermic Effects.—Besides the advantages indicated, alcohol has also another effect little looked for. One might naturally suppose that as it augments or maintains the forces, it must produce more heat which is transformable into physical energy; this is, however, not the case; it is refrigeration and not calorification which predominates. Whatever may be the dose employed, there is never hyperthermia; on the contrary when the dose is large, as Cuny Bouvier and Binz have shown in the human subject, the thermometer falls. Grobe, on giving to a healthy horse 100 grammes of rum every two hours, observed at the end of twenty hours a refrigeration of from 1.7° C. to 3.5° C.; in feverish horses the result is still more marked.

In my laboratory at the Hôtel Dieu, Demonly has noticed the necessity of administering to the dog very large doses to produce this result. In a dog weighing 10 kilogrammes (about 21 pounds), it needed 40 to 60 grammes (from 1 to 2 ounces) of brandy to lower the temperature one degree; this represents a dose of 250 to 350 grammes (8 to 12 ounces) for a healthy man, and even more in the case of a patient affected with fever, as Todd has pointed out. We may henceforth lay down this general law: the fall of heat in febricitants is proportioned to the quantity and the concentration of the alcohol; the temperature mounts up again as fast as the alcohol is eliminated. This remarkable result might have been easily foreseen; from the moment when it was understood that alcohol restrains the oxidations it was easy to account for the refrigeration.

Resume. Applications to Pneumonia.—All is involved in the physiological and therapeutical history of alcohol.

Alcohol has only incomplete alimentary properties, but it has in a higher degree the power of restraining waste, and in this way it preserves the tissue elements and their forces; moreover, as it slows or suspends oxidations, it acts in fevers as a refrigerant or antithermic. In the febricitant, alcohol is not even to be despised as an alimentary means. When other aliments can neither be digested from want of gastric juice, nor absorbed, one is always sure, as alcohol is directly absorbed without undergoing change, to find in this agent an infallible, though feeble, auxiliary to nutrition. This would be, then, a vast advantage if one could with impunity introduce a sufficient quantity; but if the amount useful for alimentation is toxic to a healthy person it is far from being as much so to a sick man,

and provided the agent be used in suitable moderation, it may render great services.

Alimentary Drinks.

I shall only mention milk, which is rarely digested; meat broth, which does not nourish but which sustains the strength; coffee, which acts exactly in the same manner as alcohol, and gelatine broth, which is also like alcohol and coffee a protective means against denutrition.

CHAPTER XXVII.

NATURIST METHOD.

Natural History of Acute Pneumonia.

Before commencing the study of the treatment of the various forms of pneumonia and submitting them to impartial criticism, we must take a survey of the natural history of the disease, that is to say, its normal evolution, and the divers individual conditions, physiological or morbid, which may influence its march, its gravity and its issue.

Regular Evolution.—In all the epochs of the history of medicine there have been found courageous and clear-sighted physicians who have not feared to stand out against dogmatists of all sorts, and leave the disease to its natural course without tormenting the patient by violent, disturbing and unseasonable medications. But the sage instructions which were left us by Van Swieten the great practitioner, Stahl the expectant animist, Borden, the worthy representative of the school of Montpellier, would have passed unheeded; their precious writings on the true clinical method would have remained a dead letter, if in recent times zealous and progressive men like Wunderlich and Traube had not succeeded by the aid of thermometric observation, which alone is capable of giving accurate notions as to the duration and end of the morbid process, in rigorously determining the natural history of the disease. We know now that pneumonia, whatever may be the treatment, follows a clearly defined typical course, a fixed cycle; that it is characterized by a sudden ascension of the temperature, which succeeding the initial chill, immediately or a few hours after, mounts up to 103° or 104° F.; that it is manifested thenceforward by a continued fever, recalling still the normal diurnal oscillations; that finally it terminates by a *crisis*, which in from six to twelve hours brings back the heat to the normal type; or else, as happens in exceptional cases, it undergoes a gradual decline, by lysis, as is especially the case in asthenic pneumonias. Here, then, is the regular type.

Duration, Crises and Critical Days.—The crisis generally occurs from the fifth to the eighth day; statistics show that it is on the seventh day that the defervescence oftenest takes place, and is less frequent on other days the farther they are removed from the seventh day. Out of 824 cases collected by Liebermeister from figures furnished by Wunderlich, Griesinger, Lebert and others, it appears that the crisis took place on the seventh day in 177, on the sixth day in 129, on the eighth day in 114, on the fifth day in 113; that is to say, in 533 out of 824 cases, or more than five eighths, crisis supervened on some one of the four days including and bordering on the seventh of the pneumonia. The crisis was also noted in 107 cases during the first three days; these were abortive cases. After the eighth day, and till the eleventh inclusive, the crisis was observed in 149 cases, then there was a small fraction representing protracted pneumo-

nia. Thus recovery pivots around the seventh day. Hippocrates indicated for all the acute diseases odd days as marking, oftener than even days, good or bad crises; the physician should wait for the advent of the following days: 3d, 5th, 7th, 11th, then 17th, 20th, in preference to all others. Traube has verified the exactness of these rules in applying the Hippocratic data, at least in the pneumonias, to the 5th, 7th, 9th and 11th days; it is then especially that your antipyretic medicines are indicated, which will act with more certainty on those days. In 18 cases of pneumonia he saw the crisis fall on odd days, and in 32 cases in which he took account of the pulse alone he noticed but once a fall in the pulse on an even day. Whether Traube's calculation comports precisely with tradition or not, is of little consequence; there are days of crisis as there are days of critical outbreaks in tertian ague (the third day), in quartan ague the fourth day, as finally happens in the case of all the eruptive fevers, which are in general of parasitic nature. Modern observation confirms the existence of the morbid cycle, and this is still another proof of the parasitic nature of pneumonia. The school of Liepzig represented by Wunderlich, insists also on the morning and even more durable remissions which may manifest themselves after the third day; it also marks certain indicative days; a lull, for instance, takes place the fourth or fifth day and all seems restored to order, but the next day the disease reappears in all its violence. There generally occurs, moreover, on the day before the crisis a recrudescence, which it is well to keep in mind; the sixth day is one of the most menacing, being the last act in the drama, the final event of which takes place the seventh day; a knowledge of this fact on the part of the medical attendant will enable him with coolness to announce the safe issue even when the symptoms seem most threatening; but to attribute the recovery to the remedies administered at the last hour would be sheer ignorance and nonsense, or knavery.

We know, whatever may be the method of treatment, that it can never cut short or abridge the duration of the disease; the cycle remains firm and unbroken.

Herpes.—There is an attendant phenomenon which is not critical, but which is looked upon as a favorable omen; this is the herpes labialis, which manifests itself the third or fourth day, and consequently precedes somewhat the crisis.[1] Out of 182 cases with herpes, 165 got well, there were only 17 deaths, that is, 9 per cent., instead of 24 per cent. the usual ratio. The physician, then, on the appearance of this rash, may generally prognosticate favorably. The following statistics of mortality will be of interest in this connection.

Mortality.—In making no distinction between methods of treatment, and taking into account only the mortuary tables of hospitals, we arrive at the conclusion that the average mortality is from 14.83 the minimum, to 24.5 the maximum; this percentage pertaining to adults only. The first, or minimum estimate, is in accordance with the statistics of Stockholm Hospital, where out of 2,710 patients between the years 1840–1855 there were 375 deaths. The second estimate sums up the mortality from pneumonia of Vienna from 1858 to 1870, where, out of 7,942 cases of this disease there were 1,944 deaths with autopsy. During these years there frequently prevailed epidemic pneumonias which varied singularly in intensity and in therapeutical results. In Paris Besnier obtained by including the

[1] Statistics of Giessler, Metzger and Sée.

broncho-pneumonias, the formidable figure of 30 to 40 per cent. in hospitals accommodating patients of all ages. We may admit 13.8 as the minimum average.

Individual Influences.—But individual circumstances, age especially, exercise a decisive influence on therapeutic results. The pneumonia of infancy almost always ends in recovery; that of the adult corresponds to the average which we have already given; the pneumonia of old age presents almost always great gravity, even when it is frank and fibrinous, that is, independent of previous diseases or morbid states; in fine, pneumonia in alcoholic patients is generally of bad prognosis.

If these diversified conditions are not kept in mind, there will be danger of criticizing with too much indulgence, or too harshly, the efforts of the physician, and his therapeutical methods. The local conditions have a minor interest; pneumonia rarely destroys life by extension of the lesion *i.e.*, by asphyxia; it brings the organism under the dominion of the microphytes; it is from their action only that the danger comes, according as the micrococcus remains limited to the lung or invades other organs.

Expectancy.

From the year 1825 Magendie, guided by a sound physiology, began to treat his pneumonic patients by diet, ptisans and an attentive patience, which alarmed his internes of the Hôtel Dieu, then devoted to the observance of traditional rules of treatment. In 1838, Becquerel, who was interne at the Hôpital des Enfants, rose up against bleeding, the danger of which was shown by statistics, and against blistering, which Guersant was practising without stint, and which was so often attended with gangrene.

But it was especially in 1849 that the revolt broke out; all the active treatments indiscriminately, tartar emetic as well as bleeding and other antiphlogistics, became at Vienna the subject of violent animadversion, which was founded on arguments which were sometimes unsound, but on statistics always exact. Dietl, under the direction of Skoda, reported the comparative results of 380 cases, treated, some by bleeding, some by tartar emetic in large doses, some by expectancy—in this class there were 189 cases;—among those treated by antiphlogistics there was a mortality of 20.7 per cent., the expectant method gave a mortality of only 7.4 per cent.

But this proportion did not always hold good. During the three following years, all cases of pneumonia, except those consecutive to fevers and Bright's disease, were treated indiscriminately by diet and refrigerant drinks such as nitre water. Out of 750 cases 681 got well, and three fourths of these between the seventh and fourteenth day. There were consequently but 69 deaths, *i.e.*, 11 per cent., which were naturally put to the account of pleuritic or endocardial complications; in 1856, however, there was a fatality, according to Mitchell, of 24 per cent., which is widely different from the startling results first announced. Every new series of statistics diminished the prestige of the expectant method; the table of mortality at Vienna in 1854 marked 20 per cent. Wunderlich, who practised expectancy with thermometer in hand, had poor success; the death rate attained an average of 23.5 per cent. Thereafter the new method encountered severe opposition in most countries.

At Edinburgh, while J. Hughes Bennett defended the views of the school of Vienna, Easton took his stand against this negative system, which had an easy way of attributing successes to sthenic epidemics,

and failures to asthenic epidemics, according as the type of the disease was believed to be toward recovery, or of necessarily grave or fatal order. There was the same unsettling of established medical tenets and the same uncertainty in the Scandinavian countries and the Netherlands. Magnus Huss, the judicious clinician, who treated pneumonia by expectancy aided by wet cups, had an average death rate of 17 per cent.; Schmidt of Copenhagen, a mortality of 22 per cent.; Bordes of Amsterdam, of 23 per cent. So far there was nothing settled or decisive, unless it be the general condemnation of blood-letting.

What took place in France? From the year 1828, the antiphlogistic method was attacked by Louis, then by Chomel and Grisolle, who adopted a mixed system without thinking of expectation. We must next come down to the year 1850; the let-alone doctrine received then an unexpected aid and impetus from homœopathy; Teissier and his pupils affirmed that they had lost but from seven to eight per cent. of their patients; Grandmotet acknowledged finally a mortality of 12.5 per cent. From this time Valleix proclaimed that pneumonia, which heretofore had possessed a bad repute, had an undoubted natural tendency toward recovery. Marrotte, considering pneumonia as a synochal disease, no longer bled, and awaited the result of the efforts of nature; Laboulbene declared plainly that pneumonia ought to be treated as a specific fever, and Charcot, in his *thèse d'agrégation* (1857), admitted expectancy in principle. From this time onward, the natural method gained ground every day, while the spoliative medication lost prestige. The first, however, continued to inspire doubts or reserves, the second left behind it regrets; the want of full confidence in expectancy expressing itself in various correctives, and in adjuvant treatments—"to assist nature"—such as a nourishing and fortifying regimen, broth, milk, tea and wine—all of which amounts to a partial surrender of the principle.

CHAPTER XXVIII.

DEBILITATING METHODS. ANTIPHLOGISTIC AND CONTRA-STIMULANT MEDICATION.

Blood-letting.

Clinical History.—Blood-letting ought to be examined from the standpoint of its clinical history, of its physiological effects, then of its therapeutic effects on the morbid principle, on the patient, and on the morbid process itself. It is only by the help of these multiple notions that we can form an impartial judgment concerning this medication, which has from all time engaged practitioners as well as the dogmatists.

Excessive Bleeding.—In reference to the treatment of pneumonia or pleurisy, which before the discovery of auscultation were confounded, Hippocrates seems to have contravened his marvellous doctrine of the *vis medicatrix naturæ* in acute diseases. He recommends to bleed in pneumonia if the fever is high, if there is pain in the side, if expiration is painful, if the patient coughs, and if the sputa are rusty, or fluid, or blood-red. Here we seem to have the characteristics of all the pneumonias, and it is a significant fact that blood-letting constitutes a method which Hippocrates regulates, it is true, according to the constitution and age of the individual, but which he pushes sometimes to syncope, according to the acuteness of the pains in the chest. During this long period of twenty-two hundred years which separate us from the school of Hippocrates, physicians without distinction of opinion, with the exception of Von Helmont, who protested in favor of his archæus, prescribed bleeding. Some, after the fashion of Galen and the Arabs, let blood without stint, even to the extent of three pounds a day, and this systematically, without taking account either of the patient or of the effects of these forced hæmorrhages. The method no longer admitted of contradiction, and the only dispute was as to the veins from which blood should be taken, whether of the sick side or the well side. Brissot, in 1512, was persecuted because he bled from the arm corresponding to the pleurisy, but he triumphed eventually, owing to the discovery of the circulation of the blood, and henceforth there was no opposition; those who were entitled the Neo-Hippocratists, as Fernel in the XVIth century, Sydenham in the XVIIth, found themselves, this once, in accord with the Galenists. A common ordinance was blood-letting to ten ounces, or half an imperial pint, twice a day; the same quantity the second, third and fourth days; total, forty to sixty ounces in the course of four days. These practitioners were the moderate men, the "naturists" of that day. What shall we say after this of the extremists, such as Botal, Riolan, Chirac, or that pontifical Sylva, physician to Voltaire (it is fortunate that the latter escaped pneumonia and his medical adviser). What shall we say still of modern lights in the profession who, after the fashion of Broussais and his disciples, bled abundantly, even

to utter prostration, who bled always, everywhere,—arteries, veins, and capillaries, and completed this spoliation by a starvation diet? The excuse of Broussais—if there be any—was his passion for his system, which he constructed with merciless logic in opposition to the dogmatists of his period. Still later, what was Bouillaud's excuse, who formulated and codified his system of repeated bleedings (*coup sur coup*), which he called the specific treatment of pneumonia? Was it the study of the blood which served as his guide? The buffy coat was considered from the time of Huxham as the sign of inflammation; Bouillaud reasoned in the same way, without knowing that this buffy coat called inflammatory is nothing but a stratum of decolorized fibrine, which coagulates with greater slowness in inflammation, and thus disengages itself from the red corpuscles of the blood. "When the coagulation is retarded by any cause whatever, and when the globules have had time to precipitate before condensation of the fibrine, the upper layer of the clot is devoid of corpuscles; it presents then a whitish coat, formed only of pure fibrine resting on a red coagulum formed of fibrine intermingled with red globules; this is the first stratification, which bears the name of buffy coat, called *phlogistic* because it appears most frequently in inflammations, but which we are sure to find also in a morbid condition quite opposite, namely in anæmia and chlorosis.[1]" Consequently the more abundant and repeated the bleedings, that is to say, the more the anæmia becomes pronounced, the more the buffy coat augments; it is then one of the most untoward signs which ought to be considered as contra-indicating phlebotomy, rather than regarded as an index of augmented phlegmasia. So much for the mistake of the clot.

Moderate Bleedings.—After these extravagances in the shedding of blood one is happy to find a little moderation in the practice of great and true clinicians like Borden, of physiologist physicians like Magendie, and of conscientious statisticians like Louis and Becquerel, who all protested in the name of truth, I add, in the name of humanity, against those violent procedures which sacrificed the patient to uphold the principle.

To-day amid the great body of professional men who are not merely sceptical with regard to bleeding, but oppose it from enlightened views, we hear now and then a voice advocating the old time practice, or rather urging certain mitigating circumstances in its favor; the latter we shall consider in the proper time and place.

Physiological Effects of Blood-Letting.

We are indebted to Marshall Hall for the first researches on the experimental physiology of blood-letting. It is only within the past twenty years that the subject may be said to have been fairly taken up again. Interesting treatises have of late been published by Warm Müller,[2] Lesser,[3] Cohnheim,[4] Volkmann,[5] Manassein,[6] Erb, Bauer etc., all cited in the memoir of Jurgensen.[7] In France experimentation on the blood has been worthily represented by Vinay[8] and by Hayem.[9]

A single loss of blood, according to Hayem, is well borne, repeated bleedings are less easily followed by restoration; copious bleedings made

[1] G. Sée, Lessons on the Blood, 1867.
[2] Christiana, 1877.
[3] Arch. für Physiol, 1878.
[4] Cohnheim, Path. Gen.
[5] Volkman, Hemodynamique.
[6] Tubingen, 1872.
[7] Allgem. Therapie, t. iv. 1880.
[8] Thèse de Concours, 1880.
[9] Recherches sur le sang, 1882.

at sufficiently wide intervals so that reparation may have already commenced at the time the vein is opened anew, are much graver; they alter especially the physiological type of the blood corpuscles, and impede the renovation of the blood.

But the results of large bleedings are not limited to this. These results relate to three orders of facts: 1. the physico-nervous effects on the circulation, *i.e.*, on the blood pressure, the pulse, the circulation, the respiration; 2. the modifications of the blood; 3. the disturbances of nutrition, which are among the most marked.

Effects on the Circulation.

Pressure.—Each bleeding, as Vinay and Arloing say, causes a fall of blood pressure, each new bleeding produces a more marked depression; Warm Müller, however, disputes the first assertion. In the dog, he says, the pressure falls when the hemorrhage has attained 3.76 per cent. of the weight of the body and then rises rapidly; a bleeding of 400 grammes (about thirteen ounces) in the human subject is without effect. There exists a sort of accommodation of the blood-vessels to the quantity of blood remaining, and equilibrium is quickly re-established; the vascular system has the power of adapting itself to its contents, so that after brief oscillations the pressure remains constant. (Warm Müller.) When, then, we hear bleeding spoken of as reducing the force of vascular pressure, we must bear in mind that this can only be accomplished at the expense of an excessive blood-letting, equivalent to a third of the whole mass of the blood. (Vinay.) A fact of this kind cannot be made applicable to man, for such bleeding would be downright murder, to say nothing of its uselessness; for why diminish the blood pressure when it is already below par, as it always is in the inflammatory fevers?

Pulse Frequency.—In general the pulse is accelerated; this is, however, not always constant (Lorain); the acceleration is in relation with the pressure, when the fall is very marked; intermittences and arrests may be noticed in these cases. (Hayem.)

Force of the Pulse.—The force of the pulse is not raised, according to Hales; the sphygmograph shows a manifest augmentation of the amplitude, which Marey attributes to the decline in the pressure. Vinay points out the relations of the amplitude with the frequency of the pulsations; the pulse becomes less ample in proportion as it is accelerated, which takes place during and after medium sized venesections. We shall see in the clinical applications what is the hard and small pulse of the *oppressio virium*, and its elevation, its ampliation under bleeding.

Form of the Pulse.—In fine, the sphygmographic tracing not only indicates the amplitude of the pulse, but its variations in form; the line of ascent is not modified; the summit is often more flat; then on the line of descent there is a wave due to the elastic reaction of the artery; but this wave, which indicates the natural dicrotism (the *pulsus bis feriens*), is more marked than in the normal state, and is a sign of weakness.

Quickness of the Circulation.—The artificial hæmorrhage diminishes singularly the quickness of the circulation. Volkmann shows that a venesection of one half per cent. of the weight of the body gives to the current a swiftness of 0.259 m. per second; a loss of 2.41 per cent. reduces the current to 0.088 m. It should be remarked, however, that the normal quickness is soon regained after the bleeding.

Cause of these Phenomena.—In all these modifications in the quickness of the circulation, in the frequency and form of the pulse, and in the blood pressure, there is only an exemplification of mechanical principles. The intervention of the vaso-motor system of nerves is not at all doubtful; these variations of pressure, etc., are not witnessed, as Warm Müller has shown, when in animals the spinal cord, vagi and sympathetic have previously been cut. Moreover Bernstein has seen, after section of the vagi, that injections of liquid into the blood, which ordinarily slow the pulse, no longer produce this effect.

Respiration.—Respiration is accelerated with the heart's action; but at the end of large hæmorrhages it is retarded. Besides the rhythm is profoundly troubled, and after copious bleedings the Cheyne Stokes type of breathing is sometimes witnessed; this has been attributed to anæmia of the medulla oblongata.

STATE OF THE BLOOD, HYDRÆMIA—HYPOGLOBULIA[1]—HYPOFIBRINOSIS.[2]

Hydræmia.—One of the first effects of bleeding is dilution of the serum (hydræmia.) This was proved by J. Davy, who saw the density fall from 1027 to 1021. Lesser sought for the cause of this dilution. He prevented the resorption of lymph by the blood, by tying the thoracic duct in a dog, and at the same time he noticed a hydræmia proportioned to the intensity of the hæmorrhage. It is possible, as Emminghauss supposes, that the proportion of lymph in the tissues augments, but it is even more certain that the parenchymata give up more water to the blood than ordinarily, and this in a very short space of time, for you will find the same watery state of the blood whether the bleeding was practised several seconds or several hours previously.

Globular Deficiency.—The influence of bleeding on the globules has been particularly studied by Hayem.

Large bleedings are followed by a decrease in the number of the corpuscles; this decrease does not arrive at its maximum till at the end of eight or nine days and lasts from ten to twenty days. When the bleeding is frequently repeated (*coup sur coup*), and a quantity of blood is abstracted equal to about one twentieth of the weight of the body, the anæmia is as marked as after larger bleedings far apart, removing blood to the amount of one fourteenth the bodily weight. This decline in the figure of the globules, however, never attains, in the dog at least, such proportions as to equal only two million globules to the cubic millimetre. The volume of the corpuscles is little modified. Manassein pretends that it is augmented; Erb has described certain large vesicular forms, but Buntzen denies the fact; all the globules whose diameter is about four thousandths of a millimetre remain in the same state as before the bleeding.

Globulins.—On the other hand, by the repetition of the bleedings, according to Buntzen, the diameter of the globules diminishes, and the number of those little corpuscles called globulins augments considerably the first few days that follow the bleeding. These are the elements described by Masius and Vanlain, by Lépine and Germont, under the name of microcytes, but Hayem denies their existence altogether.

Hæmatoblasts.—On the contrary, Hayem describes under the name of hæmatoblasts certain corpuscles which represent the first phase in the evolution of the red globules. Every loss of blood affects them, but bleed-

[1] Deficiency of globules. [2] Deficiency of fibrine.

ing is followed at a certain moment by a very marked augmentation—a double or triple increase—of these figured elements. This is called by Hayem "the hæmatoblastic crisis," and follows closely the period of minimum corpuscular richness, and is the indication of the commencement of globular reparation. A stage of formative activity then succeeds the hæmorrhage and shows us the rapid tendency to restoration.

Besides these modifications in the round elements, there is still a more important alteration pertaining to the contents of the globules, the HÆMOGLOBIN. The red corpuscles are in greater or less number decolorized, though less than in chronic anæmic conditions, having lost a large part—it may be as much as half—of their hæmoglobin. It must at the same time be noted that this diminution is not constant: it oscillates till the loss of blood attains 2.9 per cent. of the weight of the body, when the proportion of hæmoglobin falls suddenly, and the decrease long persists.[1]

The WHITE CORPUSCLES, far from following the variations of the red globules, are said constantly to increase.[2] Malassez attributes this to the experimental traumatism, and Buntzen denies the fact, which, till further researches, may be regarded as doubtful.

Reparation of the Blood.—It results from all these facts that the diminution in the number of the globules does not constitute the dominant lesion of the blood, and that its richness in coloring matter has a much greater importance. This colorimetric deficiency is conspicuous at the very time when globular restoration goes on, for this reparation can only take place through the agency of the minute intermediate forms above spoken of, the hæmatoblasts, which are slow in undergoing full development; and during their evolution the hæmatic value of the blood depreciates. (Hayem.) But this regeneration is without doubt accomplished by the colored elements of the bony marrow, the spleen and other glands. It only requires several hours to restore to the blood its volume when the bleeding does not exceed one or two per cent. of the weight of the body.[3] Whenever the bleeding amounts to from one to four per cent. of this weight, the globules demand from seven to thirty-four days for complete reparation.

Hyperinosis.—Does the blood by venesection undergo other alterations besides the hydræmia and corpuscular degradation? What becomes of the fibrine, which the physicians of former days sought to diminish by bleeding, considering the inflammation as a cause of hyperinosis? To answer this question the endeavor has been made to ascertain the quantity of fibrine in different successive portions of blood, the product of a phlebotomy. In this connection the experiments of Brüke are decisive. He found 2.24 parts of fibrine per thousand in the first portion, and only 0.68 in the fifth. It was more important, however, to know the effect of repeated bleedings on the fibrine, and here is the core of the problem: if now a day was allowed to elapse after a first venesection, the figure of fibrine in a dog was found to augment from 3.01 to 4.22 (second bleeding), and to 5.23 (third bleeding).[4] Jurgensen has noted the same fact in fasting dogs; a first bleeding gave 3.48 of fibrine, the second 4.60. So in fevers and in inflammations, where fasting is a constant concomitant, the hyperinosis becomes more and more manifest, and this is the contrary of

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[1] Lesser. [2] Henle, Remak, Moleschott. [3] Panum.
[4] In accordance with the experiments of Nasse and Sigmund Meyer.

what was once supposed. Thus it seems demonstrated that bleeding augments instead of diminishing fibrine.

Another peculiarity, no less important from the point of view of human medicine, is relative to the COAGULABILITY of the blood. In the sanguineous product of a mortal bleeding the time taken in coagulation is remarkably short, although the last portions of the blood withdrawn contain, according to Brüke, less of fibrine; the first drops from the vein or artery coagulate in twelve minutes, while successive samples require only nine minutes. Moreover, it is known that after a large hæmorrhage the clot separates rapidly from the serum, while condensation of the fibrine goes on somewhat slowly in the coagulum, giving the red globules time to precipitate themselves to the bottom of the clot; the fibrine thus disengaged from the corpuscles remains at the surface and constitutes the buffy coat.

Effects on the Organic Mutations. Fatty Degenerations.—The most remarkable effects of bleeding do not consist in the circulatory troubles nor even in the alteration of the blood. Phlebotomy determines a third order of phenomena which indicate a profound modification in the nutrition, namely, in the gaseous exchanges, in the formation of the products of oxidation, and in the fatty degeneration of organs.

The GASES of the blood undergo important changes;[1] OXYGEN is absorbed in less quantity, as the experiments of Hüffner and Panum prove. In taking for the type 100 volumes of this gas in arterial blood, the figure will be seen to fall to 87 after the second bleeding. It is noteworthy that this enfeebled absorption coincides precisely with a lessened quantity of hæmoglobin, although the number of red globules may not be appreciably lessened, as in the last three experiments of Panum. This lends support to the doctrine of Hayem, who considers the decrease in the colorimetric value of the blood as bearing relation to the imperfect globules, whose hæmoglobin has diminished, as well as to the loss of fully formed corpuscles after copious blood-letting, and the withdrawal from the economy of their functions.

According to the researches of Mathieu and Urbain, hæmorrhages occasion a no less important deficiency of CARBONIC ACID exhaled. This fact also results from the experiments of Hüffner and of Finckler, who made a careful comparative examination of arterial and venous blood after copious venesections. In fine Bauer has noticed in the starved dog a fall of twenty-three per cent. in the carbonic acid eliminated and of thirty per cent. in the oxygen absorbed.

Exaggerated Production of Urea and Uric Acid.—Besides the grave changes which are effected in the gaseous exchanges, we should emphasize the general denutrition which manifests itself by the exaggerated elimination of urea. Bauer has shown that in a dog deprived of nourishment and of water, a bleeding to the extent of from 1.2 to 3.4 per cent. of the weight of the body, determines in twenty-four hours an excess of urea and uric acid, which may attain three times the normal figure, and this inordinate elimination lasts several days; it is accompanied, as Lépine and Flavard have shown, by a still more marked excretion of phosphoric acid.

Fatty Degeneration of Organs. Fatty Accumulation.—At

[1] See the experiments of Hüffner, Mathieu and Urbain, Paul Bert and Regnard, Vinay, etc.

first thought it would seem that these blood losses must entail a diminution of the weight of the body. It is, however, not so, and such diminution as exists by the fact of the bleeding, soon ceases to be appreciable. On the other hand, all physicians and all physiologists have noticed an increased production of fat; the one class in the case of the chloro-anæmic,—which is a common observation, the other in the case of animals which have been made the subjects of several artificial hæmorrhages. Bauer, Vulpian and Dechambre have insisted on this fact, which seems to demand an explanation.

Here we find two views put forth. Bauer affirms that by reason of the loss of blood the albuminates undergo a more marked decomposition, while the exhalation of carbonic acid diminishes; this goes to show that the destruction of the fat of the economy must be lessened, whether this be derived from the food, or whether it may have been formed from the blood and deposited in the tissues, or whether it results from breaking up of the albuminates; he compares, finally, the disintegration of the albuminates to that which takes place under phosphorus poisoning. These albuminoid bodies undergo decomposition into urea, while the non-azotized substances remain in the cells under the form of fat. Hence results a true fatty degeneration.

Franckel, on the contrary, attributes all these phenomena to deficiency of absorbed oxygen. From this results the decomposition of the albuminates, which break up into more stable principles. The series is connected in the following way: the blood-letting produces the deficiency of absorbed oxygen; the deficiency causes the disintegration of the albuminates; the metamorphosed albumen is decomposed and eliminated under the form of urea; the theory, which seems quite rational, stops here; it does not explain, like the preceding, the formation of the fat. Now this fatty degeneration of the organs, and in particular of the heart, plays a considerable part in the production of the morbid phenomena which follow the hæmorrhage.

Thermometry.—Losses of blood in animals, provided they are abundant, occasion at the end of several hours a refrigeration which may amount to one degree or more, and give place later on to an elevation of temperature, due without doubt to the traumatism. In man they do not act in this way except when they are very abundant. In fevers the thermic decline is scarcely marked after blood-letting;[1] in pneumonia, Thomas, Bleuler, and Niemeyer have remarked that if the temperature falls after venesection it rises again very rapidly.

Consequences.—All these trophic mutations exercise on the secretions, on the muscles, on the heart itself, on the nervous system, the most marked effects, the study of which will better find a place in the therapeutic applications of blood-letting.

Therapeutic Effects of Blood-letting in Pneumonia.

Before pronouncing judgment on the value of blood-letting in pneumonia, we have deemed it proper to examine this ancient antiphlogistic method by the exact and impartial scientific data which experimental physiology has put at our command. We now know that abundant or repeated bleedings, such as were practised by Broussais and systematized by Bouil-

[1] Traube, Maurice, Billet.

laud, give rise to temporary troubles of the circulation, to a loss in hæmoglobin difficult of reparation, and to a profound perturbation of the movements of nutrition regarded from the point of view of the gaseous exchanges of the blood, and degeneration of the parenchymata. It is no use to argue about the difference between the effects obtained in the healthy animal or healthy man and those obtained in the patient affected with pulmonary inflammation; the difference does not exist, and we must accept these results of experimentation as entirely applicable to the therapeutics of pneumonia, the only disease in which the traditional treatment is still advocated.

If (what is impossible) clinical experience furnishes data contrary to experimental physiology, we should be made to understand; 1. the power of blood-letting to destroy the specific agent of the disease, to arrest the causes of death, and to favor the natural means of recovery; 2. the action of blood-letting on the patient; 3. its effects on the morbid process, local or general, simple or complicated, regular or exaggerated in its manifestations; in other words, what does bleeding do to the morbigenous parasites, to the patient, and to the inflammatory lesions? The last few years there has been a general renunciation of the absolute exclusive method so prevalent during the memory of many now living, when blood-letting was the sum and substance of treatment, for a very much more moderate use of the lancet, which is now reserved for special indications derived from individual conditions, from the aggravated manifestations of the morbid process, which may oppose the natural march of the disease and compromise the life of the patient. It is well to inquire if this plea of extraordinary occasion has any justification, and if there is thereby imposed on the physician the obligation to let blood; before all, we must know the general effects of these artificially provoked hæmorrhages on the causal agencies of the disease.

Effects of Bleeding on the Causes of the Disease.

How is the Pneumonic Parasite Affected by it?—The school of Cos has always considered the acute diseases as deriving their source from an undetermined principle which resides in the blood, and which the *vis medicatrix* of nature endeavors to elaborate, then eliminate by the critical secretions. In withdrawing a part of the blood, you restrain the action of the morbigenous product, at the same time that you favor the vital reactions and the salutary efforts of nature; here you have the Hippocratic dogma, which has never been lost sight of, despite the mistakes and the theories which it has sanctioned all through the ages. It was a hazardous conjecture to make, and has been fruitful of mischief.

The morbid agent in fact does not inhabit the blood, it is in the lungs. Proofs accumulate which authorize acceptance of the parasitic doctrine of pneumonia, and which enable us to signalize the seat of the micrococci in the pulmonary parenchyma, to the exclusion of every other organ, and of all the liquids of the economy. What can blood-letting do to combat the microphytes in the lungs? You might deprive the patient of all his blood without at all affecting these virulent agents in their habitat. What can blood-letting do to eliminate them? Nothing. It is not by reducing the forces, and by impoverishing the elementary tissues, by removing the blood corpuscles or their hæmoglobin that you can aid the organism to expel the parasites which have invaded the lungs. The body wastes to

repair its losses, while it needs the integrity of all its constituent elements to restrain the invasion of the micro-organisms and oppose their multiplication.

The causal indication not being clear, some zealous apologists of the old method have theorized respecting a change in the type of disease; the "medical constitution" they say has changed; the epidemic is sometimes frankly inflammatory, sometimes of a bilious, sometimes of an asthenic nature, requiring now bleeding, now emeto-cathartics or stimulants. I find nothing to support this view; certain it is that we see nothing of this markedly phlogistic "medical constitution" demanding spoliation, either in epidemic or sporadic pneumonias.

Influence of Blood-letting on the Causes of Death.—Can blood-letting affect the causes of death, so as to diminish the mortality in pneumonia below 13.8 per cent. for instance? Louis replies categorically in the negative. "Pneumonia, treated by blood-letting, has furnished a mortality of 32.5 per cent. (*i.e.*, 40 deaths out of 123). The utility of bleeding has not been more marked in cases where it has been copious and repeated than in those where it has been moderate. You cannot *jugulate* these inflammations by bleeding." After this judgment, so tersely stated and based on careful statistics, there is nothing more to say; it is evident that blood-letting, instead of saving life, increases the fatality of pneumonia in a frightful ratio. It is proper to ask what cause of death it is likely to influence, whether, for instance, it favorably modifies the asphyxia resulting from extension of the lesion? It must be borne in mind that it is not in this way that pneumonic patients ordinarily die, and if it were, bleeding is incapable of arresting the extension of the disease. When the patient dies with a limited pulmonary lesion, the death is sometimes laid to the charge of the fever; it is not, however, the fever that kills, and if this were the case, blood-letting is powerless to arrest or materially moderate the febrile movement.

Influence of Blood-letting on the Causes of Restoration, on the Duration and Crisis.—Incapable of lessening the mortality or even the chances of death, blood-letting has no power to abridge the duration of the disease. According to the method of Bouillaud, who used to prescribe the letting of from four to six pounds of blood in cases of medium gravity, and this during the first three days, the disease was expected to yield on the third day; "it is jugulated," he says, "and even when it has reached the second stage, it rarely resists beyond the fourth day."

Now we know that pneumonia, like all infectious diseases, pursues invariably a certain cycle, that defervescence does not generally manifest itself till from the fifth to the eighth day. Out of 824 cases the crisis appeared during the first three days in only 107, and these were simply abortive cases, which to-day are called congestive pneumonias, and which are frequently observed in the infant, less frequently in the adult, and still less in old age. Hence in one eighth of the cases the evolution of the disease may be spontaneously arrested in the first stage, but no kind of treatment can claim for itself this happy issue; no method has the power to abridge the course of the disease; certainly blood-letting has not this privilege.

Effects of Blood-letting on the Patient. Age and Constitution.—From all time the partisans of blood-letting have been freely disposed to make certain concessions to the age of the patients. In infants bleeding is useless; in the old man it is inefficacious; at the two extremes

of life it is dangerous; there is, then, no reserve to make except in favor of the adult, *strong* and *vigorous*, and it is in these terms that the moderate phlebotomists formulated the antiphlogistic treatment.

The uselessness of blood-letting in the YOUNG CHILD affected with true pneumonia was shown from a very early period. Alfred Becquerel, Legendre, later Rilliet and Barthez, and Cadet de Gassicourt protested against the antiphlogistic medication for a very simple reason, namely, that in early life pneumonia gets well in the immense majority of cases. Barthez noted but 2 deaths in 212 children, or scarcely 1 per cent. Ziemssen reckons 7 deaths out of 201 cases of fibrinous pneumonia in children between the ages of 1 and 16 years, *i.e.*, 3.3 per cent., and Jurgensen noted only 4 deaths out of 110 patients aged less than ten years. Therefore blood-letting has no justification in infancy and childhood, and I add, it is dangerous in this period of life when reparation of the globules and hæmoglobin is with difficulty effected. In fact among all the zealous advocates of blood-letting there is only Fernel, the neo-Hippocratist, who mercilessly spares not this tender age.

OLD MEN do not bear bleeding any better than children, and they derive no benefit from it whatever. The gravity of pneumonia goes on increasing from the age of forty, and in a rapidly progressive manner; for each decade from 40 to 80 years it exceeds 20 per cent.; between the ages of 50 and 60 it attains a figure of 50 per cent. and more. With such data before us there is nothing to hope from blood-letting, which was practised at Salpêtrière till within the last thirty years (Herrmann and Deschambre) and which Charcot now condemns absolutely.

It is, then, to ADULT LIFE that is reserved the exclusive prerogative of blood-letting. But one must not suppose that every adult between fifteen and sixty affected with pneumonia is without limitation judged a suitable person to lose even moderate amounts of blood. The few remaining partisans of blood-letting, such as my colleagues Peter and Hardy, formulate their conditions; the subject must be stout and vigorous; the disease must be of recent date (*i.e.*, not farther advanced than the first two or three days) and it must not have arrived at the stage of gray hepatization; it must not present the ataxic or adynamic form. Let us examine the individual conditions, in other words, the reservations or encouragements drawn from the strength or constitution of the patient; we shall afterwards touch upon the stage and progress of the disease and the reactions of blood-letting on the vaso-motor system of nerves.

Most medical authorities, styled classical, repeat to satiety this stereotyped phrase; "if the patient be strong, plethoric, you must bleed." How are you going to determine the strength of the patient? Is it by the force and various qualities of the pulse? If you base your indications on the feel of the pulse you are sure to go wrong; when in fact the pulse is large and bounding it is precisely then that the artery lets itself undergo distention by offering the minimum of resistance; it is then also that the sphygmograph reveals the existence of dicrotism, which is a sign of weakness in the sense that it indicates on the line of descent of the tracing, a very marked heaving of the arterial wall, a rebounding of the waves called elastic and muscular. By this it is apparent that the pulse has not the signification which has been attributed to it. At the same time there is a form of pulse which presents exactly the tracings and the appearances which are the opposite of strength; I refer to the small, hard, concentrated, scarcely perceptible pulse which ordinarily

coincides with a marked depression of the forces; it is the pulse of the *oppressio virium*. Now if, under these circumstances, you open a vein the peripheral circulation, previously enfeebled, becomes more active; the pulsations regain immediately their normal character. This, then, seems to be the vital indication for blood-letting, if there be indeed any indication, and bleeding in these conditions gives instantaneous relief; but such conditions are very infrequent and very transient; certain it is, however, that only on such an occasion does the pulse reveal the state of the forces, which are rather to be estimated from the appearance and constitution of the patient; the pulse being often an uncertain criterion.

The PLETHORIC aspect of the patient does not furnish any indication. We do not indeed know if there be a true plethora (a plethora *ad molem* as the ancients called it). We see on the animated visage of some persons certain venous varicosities which suggest an exuberance of blood; it is simply a case of blood stasis in the veinules of the face. If you bleed these supposed plethoric individuals, who are for the most part persons given to alcoholic excesses, you will see disastrous effects follow the operation. Peasants also often have those colored cheeks, that florid hue of the lips, that reddish or even livid tint which they say denotes plethora; they, however, bear bleeding badly; all country physicians are in agreement on this point.

There is the same difficulty in judging of the INDIVIDUAL CONSTITUTION and whether the patient present the characteristics of scrofula, whether he have the bilious tint, or whether he be of a nervous temperament, bleeding is no more and no less indicated than when you have to do with those psuedo-plethoric individuals or those muscular athletes who seem, according to common repute, to enjoy an excess of health. The absolute reservations to be made, the perfectly clear contra-indications of blood-letting, pertain to the anæmic, to the chlorotic, to persons exhausted by excessive drains, or worn out by immoderate labor. With respect to this entire category of patients, everywhere so numerous, especially in this neuropathic age, the interdiction of blood-letting is rigorously imperative.

Constitution of the Gauls.—Has it perchance happened that the native French constitution has deteriorated—become feminized and anæmiated since the commencement of this century? This is what the old partisans of blood-letting have maintained when they have seen themselves abandoned by physicians and by patients. Galen, who was prodigal in blood-letting, was very sparing of the Gauls. "Proinde minus detrahimus quod ad corporis habitus, in candidis et quibus mollis teneraque est caro, quales Galli sunt." ("Hence, as touching habits of body, we remove less blood from persons of pale complexion, and from those whose flesh is soft and tender, among whom are the Gauls.") Behold us then! again become the Gauls of the time of Galen, after having been the athletes of Chiràc, Broussais and Bouillaud.

Effects of Blood-letting on the Lesion.—Abstraction being made of the causal agent which constitutes the *raison d'être*, I had almost said the personality of pneumonia, I find but one lesion, and this is a collective name to designate: 1. the anatomical lesion of the lung; 2. the chemical lesion of the blood; 3. the alterations of the tissues (oxidation, fever); 4. the histological and functional modifications of the heart; 5., finally, the nervous perturbations, which have nothing about them which is essential. There can be no pneumonia without the inflammatory processes designated under the denominations of engorgement, red or gray

hepatization; no lobar pneumonia without hyperinosis of the blood, without fever, and perhaps without adynamia of the heart. Let us examine these different morbid elements, and take account in each case of what blood-letting can do to attenuate them or reduce them to naught.

Anatomical Lesions. Nature of the Pulmonary Engorgement.—In the first stage, pneumonia consists in a subsidence of the pulmonary tissue, which ceases to be aëriferous; the capillaries are all enormously dilated, the alveoli are in part filled with red globules, and an exudation, still liquid, which has already modified some of the endothelial cells. This alteration, which is also met with in œdema of the lungs (Friedländer), is not characteristic of the inflammation, and according to the judicious remark of Lépine, it is the prelude of the phlegmasia.

Effects of Blood-letting on the Pulmonary Engorgement.—Now, all clinicians who advocate bleeding are agreed in recommending it especially or even exclusively in this preparatory period, which may last two or three days, and which is characterized during life by a sanguinolent expectoration and crepitant râles, without souffle or dullness. One or two opportune bleedings, are sufficient, they say, to dissipate this engorgement and jugulate the malady. It is certain that one sees now and then pneumonias which are arrested at this stage, and this without any active interference on the part of the physician: they are in fact such pneumonias as abort before the inflammation proper is established, before the hepatization. But what can bleeding do to favor this arrest, and especially to transform all pneumonias into abortive forms? In clearing the vascular system, in diminishing the mass of blood, blood-letting without doubt facilitates the general circulation in the sense that it provokes a temporary diminution of the intra-vascular pressure. Now we know that every pre-inflammatory congestion, which differs altogether from functional hyperæmia, consists above all in vascular dilatation, in a slowing of the course of the blood and in a diapedesis of the white and red globules which can only take place by reason of this slowing and of these dilatations of the alveolar bronchial capillaries.

Can any one believe that the general weakening in tone of the blood-vessels which follows bleeding is going to remove the excess of blood stagnating in the lesser circulation, prevent the ectasy of the broncho-pulmonary vessels, and in this way, the transudation of red and white corpuscles, the formation of the exudation, and above all the degeneration and shedding of the endothelial cells?[1] The relation of the physiological action of blood-letting to the pre-inflammatory state is impossible to define; its therapeutic action is problematical.

Red Hepatization. Gray Hepatization.—In the second stage there forms in the pulmonary alveoli and in the terminal bronchioles, a co-

[1] Every inflammatory process, besides the alteration of the blood-vessels, the retardation of the circulation, the diapedesis of leucocytes, the sero-fibrinous exudation, comprehends a series of degenerations of the parenchymatous cells, beginning in a cloudy state, called necrosis by coagulation, and undergoing fatty or cellulo-fibrous transformation.

Functional hyperæmia, which characterizes an organ engaged in its physiological work, differs chiefly from inflammatory hyperæmia in the alteration of the walls of the blood-vessels (the vascular ectasy would of itself rather accelerate than retard the blood current), whence results the stasis and the diapedesis. The condition of the vaso-motor and trophic nerves of the organ is undoubtedly widely different in the two instances. (See the Author's Work on "Phtisie Bacillaire," page 562.—Trans.

agulable fibro-albuminous exudation, which fills these spaces to the extent of one or several lobes, gives then the consistency of liver (whence the name hepatization), and manifests itself at the surface of section under the form of little red granular prominences due to these coagula. The alveoli, filled with this fibrinous exudation, have lost their epithelium, and where vestiges of the epithelial lining remain, you notice this circumstance by augmentation of volume and proliferation of the cells.

From this moment the morbid process tends towards restoration or towards death. If recovery be the issue, the red globules extravasated at the onset, dissolve, and the fibrinous exudation (probably by the intervention of a peptic ferment,) liquefies and undergoes resorption at the same time as the fattily degenerated epithelium. If the march of the disease be toward a graver issue, the hepatization changes from red to gray, that is to say, the red globules are replaced by leucocytes; the exudation becomes more abundant, lactescent, purulent; this is the third stage, or period of purulent infiltration. When the lesion has passed through the first stage to that of the inflammation proper, the partisans of the antiphlogistic treatment do not at all abandon blood-letting. In fact the hepatization may show itself as such from the very onset; the tubular souffle and the dullness which characterize it are often perceived from the first day, without there having been a single crepitant râle. These cases are believed to be amenable to the same treatment as the congestive stage, and it is supposed that the inflammation may be arrested in its downward tendency. But in any event, whether the hepatization be such from the commencement, or follow the ordinary course,—how is such a miracle to be effected? Is it by preventing propagation of the inflammation to the neighboring parenchyma? Such a result cannot be comprehended as possible. Is it by opposing the development of the pre-inflammatory congestion? We already know the impotence of blood-letting to effect this. Is it, finally, in promoting the resorption of the exudation? Since the famous experiments of Magendie on the rapidity of absorption of poisons after bleeding, the action of bleeding has been interpreted in the sense of favoring the resorption of exudations. There is, however, a serious difficulty in the way of acceptance of this view; the fibrinous exudations intermingled with epithelia cannot enter the circulating current till after having undergone a preliminary softening and liquefaction, and there is no power in blood-letting to hasten this transformation. Hence on whatever side we seek for the benefits of blood-letting and its influence on the anatomical lesions, we meet with nothing but obscurity, hypotheses and mistakes.

Chemical Lesions of the Blood. Hyperinosis.—The ancient school of Vienna placed the source of all inflammation, particularly of the fibrinous exudation of pneumonia, in a special crasis of blood characterized by excess of fibrine; this state was called *hyperinosis*, which proved to be a lucky word, being eagerly adopted, even in France, doubtless because the fact of excess of fibrine is scientifically true. It was demonstrated more than forty years ago by Andral and Gavarret (who proved it by processes of weighing) that the fibrine, which normally oscillates between $2\frac{1}{2}$ and $3\frac{1}{2}$ parts in a thousand, mounts up in pneumonia to 5.6 and even 9 per 1000, but these great experimenters never concluded on this account that this state of the blood was the cause of the phlegmasia; it is only the positive and constant voucher of inflammations which suddenly make their appearance in the midst of health.

Defibrination by Blood-letting.—Logically it should be shown that blood-letting, so vaunted as a remedy for this hyperinosis, has the power to defibrinate the blood; but it has been found (as before said) that it is precisely the reverse which takes place; the fibrine after successive bleedings, more or less near together, undergoes positive augmentation, as in the case of anæmia generally, so that the assertion that bleeding diminishes the hyperinosis has proved to be a baseless assumption.

Another effect of bleeding is to LESSEN, at least temporarily, the number of RED GLOBULES, to augment the *globulins* and diminish the HÆMOGLOBIN. Now, pneumonia is a disease which itself impoverishes the blood, without doubt much less than acute articular rheumatism, but enough nevertheless to contraindicate the augmentation of this state of impoverishment by blood-letting.

Blood-letting can effect nothing against the FEBRILE TEMPERATURE; its refrigerant action is much disputed, and is always very insignificant and transient.

The OXIDATIONS which are produced during and by the pneumonic fever, are manifestly augmented, as the excess of urea in the urine testifies; blood-letting gives rise to identically the same phenomena, so that denutrition is promoted at the same time by the disease and by the remedy.

THE FATTY DEGENERATIONS are equally the result of the fever and of blood losses; one of the best proved consequences of the hyperthermia is the disintegration of the muscles, the fatty metamorphosis of the spleen and of the liver; sanguineous emissions also produce fatty accumulations of a deplorable kind, and this at the expense of the healthy performance of the functions of the organs affected, particularly the heart.

There remains to mention the action of blood-letting on the HEART, which is either degenerated or enfeebled, and on the BLOOD-VESSELS, whose tension is already diminished by the fever. It is needless to say that bleeding may indeed augment these grave perturbations, but it is incapable of modifying them.

Aggravation of Pulmonary Lesions.—There is only one circumstance that seems at all to justify a moderate phlebotomy in pneumonia; it is in that relatively infrequent condition of aggravation of the pulmonary lesions attended with œdema of the interstitial tissue and active collateral congestions; but this state will be discussed farther on under the head of complications. Practically it may be said that we are done with the antiphlogistic régime in this disease.

CHAPTER XXIX.

CONTRA-STIMULANTS, ANTIMONY.

History, Glorification and Condemnation of Antimony.

Known under the name of stibium and used for cosmetic purposes, the black sulphuret of antimony was employed, according to Pliny, by the women of Asia to blacken their eyes. It is commonly said that the chemist and monk, Basil Valentine, poisoned a number of his brethren by too freely purging them with this metal, whence the name by which it was known in France—*antimoine*. It is, however, to Paracelsus that we are indebted for first bringing antimonial medicines into vogue, and the marvellous properties of the stibian preparations were vaunted by this same Basil Valentine in a book which appeared in 1606 under the singular title of *the triumphal chariot of antimony*.

The number of antimonial preparations known and discussed, was at first fixed at twenty-one; each had the proper number of its rank: then only nineteen were admitted, and the figure was soon reduced to five, to wit, the three sulphides of antimony, which were more and more rarely used, the chloride, which was employed as caustic, and the famous potassio-tartrate, or tartar emetic. To-day only the red sulphide is known (Kermes mineral), the golden sulphide, the oxide and tartar emetic; all the other stibian compounds are sunk in oblivion, after having had their day. Condemned formally in 1556 by the Faculty of Medicine at Paris, backed by an edict of Parliament which condemned antimony as a poison and prohibited its use, this substance became, in the hands of Joseph Quercitanus in 1575 the great purifier of the blood, then, ten years later, the anti-dyspnœic remedy of Rembert Dodonæus. When in 1631 Adrien de Mynsicht of Mechlenburg discovered the soluble preparation, the familiar emetic compound with potassa, the renown of the antimonials was restored in full splendor, and the Faculty rescinded the original decree against the remedy by a large majority (92 out of 102 voting).

In the eighteenth century this medicine came greatly into vogue as an emetic, and as such it was largely prescribed; not by Baillon, who was already dead, not by Lazarus Rivierus, whose academic début was so painful, nor by Baglivi, the philosophic medical writer, but by Stoll, who chanted the praises of tartar emetic in the treatment of all the pneumonias, which he qualified with the appellation of bilious, in order to justify the emetic treatment. But this was not all.

A still grander destiny seemed to await tartar emetic when at the end of the last century, to its already marvellous properties was added that of opposing the diathesis of *stimulus*, a hypothesis brought into repute in Italy by Rasori in opposition to the theory of a *sub-stimulus* diathesis, proclaimed in Scotland by Brown. While the latter urged the necessity of a supporting regimen against the lessened stimulus, the former, who taught the doctrine of hyper-excitation, announced the contra-stimulant powers of tartar emetic,

and showed the tolerance of this medicament in massive doses. It was henceforth, the true, the only curative means in all maladies marked by excess of excitation. Contra-stimulism and its agent of predilection rapidly gained prestige; Laennec, Chomel, Louis, Grisolle adopted this practice, and tartar emetic, unfortunately, still dominates the therapeutics of many diseases where it would be an advantage to dispense with it altogether.

Doses and Modes of Administration of the Insoluble Antimonials, of Tartar Emetic in Emetic and Contra-stimulant Doses.

The insoluble antimonial preparations comprise Kermes mineral, the yellow or golden sulphuret, and the white oxide; tartar emetic is alone soluble in any dose. Kermes is a hydrated oxysulphuret of antimony which can only be administered suspended in syrup or mucilage, or in pastilles. The golden sulphuret is a penta-sulphide (antimonic sulphide, $Sb\ S^5$) mixed with a little oxide. The white oxide, brought again into vogue by Trousseau, is not an oxide but a biantimoniate of potassa, which also bears the name of *washed diaphoretic antimony*. Kermes is prescribed in the dose of 5 to 15 centigrammes ($\frac{5}{6}$th gr. to $2\frac{1}{2}$ gr.): the white oxide has been given in doses of from 1 to 10 grammes (15 to 150 grains) and more; tartar emetic is emetic in the dose of 5 centigrammes ($\frac{5}{6}$th gr.); in doses of from 30 centigrammes to 1 gramme (5 to 15 grains) it is administered as a contra-stimulant. The doses of the insoluble preparations above given have no special significance, for in order not to obtain an emetic effect they must all be administered in fractional doses wide apart; what is demanded of them is a nauseant and expectorant action. Kermes is ordinarily taken in doses of only one centigramme ($\frac{1}{6}$th gr.) at a time, and the potion which contains 10 to 15 centigrammes ($1\frac{2}{3}$ to $2\frac{1}{2}$ gr.) is distributed through the twenty-four hours. It is the same with the white oxide, which is given in small doses and frequently repeated; thus a mixture containing 2 to 3 grammes (30 to 45 grains), is exhibited in teaspoonful doses every half hour or so, so distributed that the whole shall be taken during the day. This is also the rule with tartar emetic, if you would avoid vomiting, and cause large quantities to penetrate the economy.

Easy Absorption of Antimony by the Organism. Comparison with Arsenic.—Antimony, from a physiological as well as from a chemical point of view, is classed by the side of arsenic in the group of the heavier metals; both, however, differ from the other metals by their feeble basic action, that is to say, by the acid character of their combinations with oxygen, and this circumstance is in harmony with their action on the economy.

In respect to both these substances there is this characteristic, that they are absorbed much more easily and rapidly than the metals in general, than iron and mercury, for instance; this is why they act through the blood, and from a therapeutic point of view one may depend on their promptitude of action.

Certain phenomena of acute antimonial poisoning, formerly attributed to a local irritation of the digestive organs, are now recognized as the result of a general toxic action. Hence it is that observers are often astonished that toxic and fatal doses of antimony have produced only traces of inflammation of the stomach and small intestines, while the colon was violently affected and even sphacelated; the reason is that the metal is eliminated

by the large intestine, where the stomach has not expelled it by vomiting at the time of ingestion.

Absorption by the Skin and Mucous Membranes. Local Action.—Antimony presents also an interesting peculiarity from the point of view of its absorption by the skin or mucous membranes, and of its local action. Several antimonial compounds, and among them the golden sulphide, kermes and the white oxide, are insoluble in water, and those that are soluble, as tartar emetic, are of relatively feeble stability. The first cannot act unless they are decomposed and rendered soluble by certain chemical substances which they meet in the economy, and which always act imperfectly, and this is why these preparations present a composition and an action that are inconstant; tartar emetic makes an exception. This is, however, a double salt, and like all the other double salts of the heavy metals, has the property of not combining with the albuminates when it is in a neutral or alkaline state. On the other hand, in presence of dilute acids it forms with albumen a precipitate still badly defined; nevertheless this circumstance merits consideration because the conditions which preside over this reaction are found in certain organs, and especially the stomach and skin; it is there, in the midst of acid media, that one often meets with the local lesions, elsewhere little marked.

Tartar emetic incorporated into an ointment, and rubbed over the INTEGUMENT produces papules, the pustules which are due to penetration and inflammation of the follicles of the skin, in which moreover there exists an acid.

Introduced by the MOUTH, tartar emetic may determine pustulation of the buccal, guttural, œsophageal and even gastric mucous membranes. This accident rarely follows a single dose of the antimonial; there is not even inflammation, there is only an epithelial desquamation (Hanfield Jones); but if the administration of the remedy is continued, even in small doses, little ulcerations may result.[1]

PHYSIOLOGICAL EFFECTS OF ANTIMONY.

Emetic Action.—Tatrar emetic, in the dose of 1 to 10 centigrammes (g. $\frac{1}{6}$ to gr. jss.) introduced into the stomach always ends in penetrating the circulation in some quantity, although it may be in part rejected by vomiting or by purging. When injected under the skin or into the veins of animals, it acts on the stomach as though it had been introduced there directly. It is in fact eliminated from the blood by the gastric mucosa, by the bile and by the intestines. The elimination continues by the urine, which is often loaded with albumen, but the metal is often found weeks and months afterward in certain organs such as the liver and bones. So both directly and indirectly the stomach is implicated. In fact in their personal experiences, Meyerhœfer and Nobiling always noticed, after a certain malaise and chills, an afflux of saliva in the mouth, an ardent thirst, a sensation of twinging in the stomach, then after larger doses, nausea, and after ten or twenty minutes, vomiting with griping, loose discharges, often also with great prostration of the general forces and depression of the heart's action.

Mechanism of the Vomiting.—Two opinions are prevalent as to the mechanism of the vomiting. According to the first theory it is a reflex action, due to irritation of the mucous membrane by the antimony; accord-

[1] Nobiling.

ing to the second it is the direct effect of the emetic on certain co-ordinating centres situated in the medulla oblongata. In favor of this last view, we may refer to the fact that the vomiting does not begin till at the end of a certain time, being somewhat tardy in its appearance, and on the other hand, when the antimonial is injected directly into the blood it produces the same effect as when introduced by way of the stomach. But according to Radziejewsky [1] as well as according to Kleimann and Simmonowitsch [2] the vomiting takes place more quickly and by smaller doses when the emetic is swallowed, than when it is injected into the veins or cellular tissue. Herrmann and Grimm have made the same remark; it seems, then, probable that the action of the salt of antimony is, so to speak, of peripheral nature altogether, and that it produces a special excitation of the walls of the stomach and of the terminal vagi which causes the vomiting by reflex influence. It is even probable that in injecting the antimonial into the blood it acts only by way of *return*, that is to say at the moment when the salt after being absorbed, is eliminated by the stomach; it is certain that when the emetic is injected into the veins, it is found in large part in the vomited matters.

This fact is not contradicted by the famous experiment of Magendie, who obtained retching by introducing tartar emetic into the blood-vessels of animals deprived of their stomach; this may have been effected through the influence of the emetic on the nerve terminations of the mucous membrane of the throat and œsophagus, whose nerves are equally capable of provoking the act of vomiting by reflex excitation. It is true that Gianuzzi after having sectioned the cervical cord in the dog, did not succeed in inducing vomiting by means of tartar emetic; he concludes from this that vomiting is an act of the central nervous system and not a reflex phenomenon of gastric (*i.e.* peripheral) origin; but severe nervous perturbations, often even the simple immobilization of the animal, and especially the artificial respiration necessitated by the section of the cord, suffice to prevent all possibility of vomiting, even by the most energetic emetics. We have seen, and shall again see further on, that a similar phenomenon takes place in the human subject; considerable doses of tartar emetic fail to cause vomiting in some morbid conditions, such as pneumonia and tetanus. This is called tolerance, and physicians have sought to obtain it in the treatment of these grave affections; we know that the functionating of the sensory nerves of the stomach in such circumstances is no longer possible.

The conclusion is this: The emetic always causes vomiting in a state of absolute or relative health; it always produces this effect by the intermediation of the stomach, whether the poison may have been swallowed or injected into the blood.

Nauseant Action of the Antimonials.—The insoluble preparations of antimony act in a more obscure manner on the stomach. Kermes often contains by decomposition a certain quantity of oxide of antimony, to which it owes in part its properties. Possibly a small part of the kermes ingested undergoes solution in the acids of the stomach and is thus absorbed; according to this hypothesis this compound is similar in its action to tartar emetic, but the differences are such that even with large doses you obtain but feeble effects; hence this preparation is used in practice only to provoke nausea and exert an expectorant action. It is the

[1] Arch. de Phys., 1871.

[2] Arch. de Pflüger, 1872.

same with the golden sulphuret and the white oxide. This state of nausea constitutes a sort of complex condition which is characterized subjectively by a sentiment of malaise, of sinking, of fatigue and of muscular resolution; clinically it manifests itself by an acceleration of the heart accompanied by a small rapid pulse, in all probability produced by an excitation of the accelerator nerves, considering that the blood pressure, far from rising, tends rather to undergo a slight fall. This double effect of the antimonials on the muscular system and on the heart merits a study so much the more minute as the paralyzing property which affects the entire muscular system, including the myocardium, has not only been considered as peculiar to the greater part of emetics, but has served as the basis of the entire Rasorian doctrine. It must be remarked, however, that the paralyzing action is not in direct proportion to the emetic or nauseating action, and that it may even be entirely independent of it.[1] Thus it is that apomorphia does not in any way paralyze the heart, while constituting a very efficacious emetic; these facts will be discussed at more length under the head of the soluble preparations administered in contra-stimulant doses.

Secretory Properties of the Antimonials. Purgative Effects.—What is better demonstrated is that the nauseating medicaments determine hyper-secretion, such as an increased flow of saliva and of mucus from the alimentary passages. Their effects on the intestines are not doubtful; if you administer from five to ten centigrammes ($\frac{5}{6}$ gr. to $1\frac{2}{3}$ gr.) of tartar emetic dissolved in a quart of water, there results a purgative effect which was formerly much utilized by surgeons when they wished to produce thorough evacuations. In these conditions the emetic being rapidly expelled from the stomach reaches the intestine, on which it acts by reducing vascular tone and augmenting secretion, thus favoring copious discharges.

The action of antimony on the intestines is never so pronounced as that of arsenic, which acts less on the stomach and more on the intestine, paralyzing its vessels; the vascular dilatation which is due to paralysis of the great splanchnic, the principal vaso-motor nerve of the intestinal arteries, perfectly explains the venous hyperæmia which is found in all the viscera of the abdomen.

Astheno-cardiac and other Properties of the Antimonials.

The school of contra-stimulism, certainly aimed to affect the heart by their large doses of tartar emetic, and Giacomini, the therapeutist of this school, called it a *cardiaco-vascular asthenisant.* It is a fact that the heart is smitten both in its texture and functions.

The contra-stimulant destroys the excitability, often in an irremediable manner, provided that the heart was previously feeble. One half gramme (seven grains) of tartar emetic is sometimes sufficient to accomplish this in a well man; we have seen why it is that the sick man, and especially he who is laboring under a grave attack, supports better the remedy, and in larger doses.

In cold blooded animals five-sixths of a grain injected under the skin determines, after a brief acceleration, a marked diminution in the force and number of the beatings of the heart.

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[1] Harnack.

In the dog, the force of the heart, and the intra-vascular pressure, diminish in an immediate and very marked manner. The number of pulsations, after a slight augmentation, falls continuously, and finally the contractions of the heart become irregular, and this organ, when the dose is toxic, is arrested in diastole. In the human subject a similar series of phenomena is observed in the period of nausea; at first the frequency of the pulse rises by forty pulsations; then it commences to grow slower. Then in the period of reaction, as soon as the vomiting is over, the force of the cardiac impulse and the number of the pulsations are soon re-established.

Mechanism of the Cardiaco-vascular Enfeeblement.—Several hypotheses have been put forth as to the mechanism of this cardiac asthenia. One attributes to the antimony a direct action on the cardiac muscle, as has been observed in respect to veratrine, which is similar to it from the point of view of its effects on the heart. Another considers the cardiac asthenia as the result of fatty degeneration by antimony, which is in fact a veritable steatogenous poison to the glands and muscles. A third theory supposes the intervention of a reflex nervous action of gastric origin; but one may well ask how the excitation of the gastric vagus can act on the cardiac vagus, which only manifests its irritation by slowing and not debilitation of the heart? The true theory has been indicated and recently proved by Solowejtsch.[1] On injecting tartar emetic under the skin of a frog, one may note, after several attacks of vomiting, that the reflex centres of the spinal cord are paralyzed. In the heart the inexcitability affects at first the automatic centres, and this peculiarity is common to antimony and to arsenic; finally the excitability of the muscle may be extinguished. It is this depression of the heart's action which has always been utilized in the treatment of pneumonias and of other grave diseases; but do not forget that this may be realized at the expense of the most dangerous collapse, particularly in children.

At the same time that the heart loses its energy, and even before the INTRA-VASCULAR PRESSURE diminishes, the vessels dilate, and their nerves lose their influence. It is true that this depression is but feeble, and of transient duration; what proves this is that if you bring the heart under the influence of digitalis, thus raising its energy, the arteries also recover their tension in a marked degree.

There is at the same time produced a decline in the TEMPERATURE, which in certain experiments of Radziejewski fell six degrees.

The NERVO-MUSCULAR system is severely affected. The voluntary muscles, and perhaps also the muscles of organic life, undergo a considerable diminution in their energy. The most vigorous animals which have been poisoned by antimony, are immediately smitten with the utmost prostration and enfeeblement; they can hardly take a few staggering steps without falling over on one side; the muscles are at the same time taken with fibrillary tremblings. When the vomiting is over, the condition seems momentarily to improve, but the toxic symptoms soon return with new force. One cannot but infer that the muscle is affected directly by the poison, for in the frog the curve of the contractions shows an extraordinary depression. (Buchheim.) It is certainly the antimony that works this mischief and not the potassa of the compound, as Nobili believed. Robert has proved that antimony is a paralyzer of the muscles, but that it does not act except in large doses.

[1] Arch. f. exp. Path. 1. 12.

The NERVOUS SYSTEM itself is equally smitten by the poison, and this takes place through the intermediation of the blood. There is something more here than a simple trouble of the circulation; the action of the antimony is directly exercised on the nervous substance. The proof is this: in cold-blooded animals whose nervous system is, so to speak, independent of the circulation, there is observed a paralysis of the cerebro-spinal centre and a complete extinction of the excito-motor power of the spinal cord; the reflex centres are paralyzed to such an extent that the diastolic arc cannot complete itself across the cord, as is clearly proved by the experiments of Solowejtsch. This is a fact of importance in the interpretation of the phenomenon called tolerance, in virtue of which patients gravely affected with pneumonia support large doses of the emetic, not with impunity—far from it—but without vomiting, which is a reflex act rendered impossible.

Action on the Respiration.—Antimony acts in two ways on the respiration; in disturbing its rhythm, and in modifying the secretions, especially in the pathological state. It has an expectorant action which will be alluded to when we come to the so-called bilious pneumonias. By the vomiting, and even by the state of nausea, the respiration becomes accelerated, superficial, irregular; then it undergoes retardation: then inspiration is panting and painful; expiration is labored and slow. These phenomena have their reflex origin in the stomach, for all emetics and the act of vomiting trouble the respiratory act; but there is also a direct action of the antimony on the nervous centres concerned in the respiratory function.

It has been claimed that antimony has a *resolvent* effect on the pulmonary parenchyma; this is only an hypothesis. Others have affirmed that antimony hepatizes the lung; this is not so, for out of twenty necropsies of dogs poisoned by tartar emetic, Ackerman never once found the least alteration in the respiratory organs.

Action in Favoring Degenerations.—Like arsenic and phosphorus, antimony exercises on the parenchyma an irritant action of undetermined nature, but incontestable, which depends neither on the affinity of the metal for the albuminates, nor on the precipitation of albuminates in solution; this last circumstance, as we have before said, is never realized unless tartar emetic meets with acidified albuminous solutions. All these metals are *steatogenous*, and produce fatty degeneration of the different organs, especially the liver and *heart muscle*. All, finally, in destroying the organs, determine the production of an *excess of urea*. In a dog submitted to fasting and dosed with tartar emetic, the balance between the ingestion and excretion of nitrogen having been well determined, Gaetgens found a considerable augmentation in the production and elimination of urea. All these facts agree in demonstrating the grave alterations which antimony, like other violent metallic poisons, produces in the organism.

THERAPEUTIC PROPERTIES OF THE ANTIMONIALS.

The antimonials present three therapeutic properties: they act as *nauseants*, and as *expectorants;* this is what is observed especially in the case of the insoluble preparations, such as kermes, and it is this complex action which is utilized in bronchitis. (See next chapter under the head *Emeto-cathartics.*) Tartar emetic, the only soluble salt, acts in small doses as nauseant, expectorant and emetic; in large doses it is the type of *contra-stimulants.*

Results of the Contra-stimulant Medication.

To make tartar emetic tolerated, it has been associated with a certain quantity of opium, designed to correct the emetic effect, and in this form it is given in doses of from one to two grains every two hours till fifteen or twenty grains have been taken in the twenty-four hours. The founder of contra-stimulism in this way arrived at enormous doses amounting to thirty or forty grains a day regardless of the collapse, often fatal, which is sure to follow. His followers Laennec, Grisolle, Trousseau, stopped at ten or fifteen grains; some ordered nothing more, while others commenced by the traditional bleeding which they had not the courage to proscribe absolutely. Now see the results: out of 648 patients of the city hospital Giacomini counted 22 per cent. of deaths; and out of 180 military patients, 14.5 per cent. of deaths. Grisolle had 6 fatal cases out of 44 patients of medium age of 37; this was the method pure; combined with blood-letting it gave a mortality of 10 in 80. Finally, employed at a later epoch, which no longer permitted recourse to blood-letting, it furnished the enormous number of 18 deaths out of 30 cases. What more striking condemnation of the system could be asked?

Danger of Empoisonment.—It is demanded with some reason, if, in the causation of this frightful mortality, the stibian poisoning does not enter for a large part. It is certain that many patients experience incoercible vomiting and diarrhœa, and die of stibian cholera; in others the prostration becomes extreme and lipothymic, the pulse filiform and irregular, the respiration superficial and precipitate; cold sweats appear with cyanosis and often a mortal syncope. To avert the dangers of the emeto-cathartic action, which used often to supervene in the midst of the tolerance, Krimer was in the habit of associating with the antimonial, cherry laurel water; in France the syrup diacode (poppy syrup) was added; all these precautions are, however, vain. To prevent the collapse, Grisolle and Bucquoi would never exceed five to seven grains a day, and they used to prescribe wine to correct the prostrating effects of the remedy. All these correctives did not suffice to stay the protestations of Gendrin, Dance and Andral in France, of Strambio in Italy, and the greater part of foreign physicians.

Restrictions of the Method.—Just as in the case of blood-letting, the advocates of the contra-stimulant method have reserved it for certain cases of pneumonia of medium intensity, for the first periods of the disease, for the pneumonias of childhood (Blacke), of the adolescent and the adult, for vigorous patients generally, while specifying as contra-indicating it, the advanced stages of the disease, old age and previous debility. This method has, however, at last met with general condemnation, and to certain timid partisans who remain, it is no longer but the shadow of the ancient contra-stimulism. The most that can be said in its favor is that with certain prudent and experienced practitioners (like Jaccoud), tartar emetic is deemed worthy of an honorable mention alongside of bleeding and cupping, and under the title of a symptomatic remedy to combat the exaggerated febrile dyspnœa.

CHAPTER XXX.

TREATMENT OF GRAVE OR COMPLICATED PNEUMONIAS.

This collective title comprehends: The cerebral pneumonias, (pneumonias with delirium, convulsions, coma, meningitis, etc.) The pseudo-typhoid or ataxo-adynamic pneumonias; The bilious pneumonias; Pneumonias marked by an exaggeration of local lesions, or of symptoms; Pneumonias complicated by grave localizations in other organs.

Cerebral Pneumonias.

In the adult and in the aged patient it is the delirious form which oftenest presents itself; in the young child we generally notice a tendency to coma and convulsions; at all ages there may supervene inflammatory meningeal complications which are less rare than one would suppose.

Pneumonias of Delirious Form.—When you notice delirium in connection with pneumonia, whether at the onset before the local manifestations are apparent, or whether in the course of the disease, the first duty is to inquire if the frenzy be of toxic nature and due to alcohol. It is in fact alcoholism which is in these cases the most common cause of delirious conceptions, or more properly speaking, of hallucinations. If you are able to exclude the presumption of alcoholism, the next inquiry should be whether the delirium may not be simply pyrexial, or whether it be not the result of inanition or cerebral anæmia, often difficult to recognize from want of knowledge of the antecedents of the patient. Lastly, in certain neurotic patients, and especially in females, there is often a delirium of nervous nature, more easy to diagnosticate. As for delirium symptomatic of cerebral or meningeal congestion, authorities have described it in connection more especially with pneumonias of the apex; such a complication would be attended with great danger, and the treatment of the pneumonia would be merged into the treatment of the encephalic inflammation. There is, however, nothing about the nature of pneumonia itself that predisposes to cerebral determinations.

Treatment of Pneumonia with Alcoholic Delirium.

The delirium breaks out with the fever. An individual whose antecedents and habits of life are unknown to the medical attendant, and who may have been unconsciously to himself drifting into inebriety (I have seen numerous instances of this kind), finds himself all at once taken with a violent fever and a delirium of a special kind. His erroneous conceptions are characterized by certain peculiar hallucinations of sight and of hearing, which lead to a sort of logical delirium, moulded, as Fournier says, on these false perceptions, and followed often by acts of fatal violence. During periods of calm the restless agitation under which he labors, manifests itself by muscular trembling. Now in presence of a scene of this

kind, do not conclude that you have before you a case of acute frenzy, or cerebral congestion; it is clearly delirium tremens with which you have to do, and this is not all;—the disease has made its appearance on the occasion of an attack of pneumonia, and if you examine for the latter you will find it, unless it be of central localization.

At other times the pneumonia already exists when the alcoholic delirium comes on; the latter lasts three or four days and generally terminates by a refreshing sleep.

This is the ordinary history of the attack; what, now, are the indications and contra-indications?

Contra-indications.—It is the absolute duty of the physician to avoid blood-letting. One hundred and fifty years ago Van Swieten pronounced a formal anathema against bleeding in the case of drunkards in general, and delirious alcoholic patients in particular, suffering from pneumonia. All accurate observers, such as Chomel, Grisolle, have sanctioned this absolute proscription. Ball adds to this prohibition the positive interdiction of the revulsive method designed to combat a cerebral congestion and inflammation purely imaginary, a method of treatment which in reality only serves to exasperate the patients and redouble their agitation. I complete the list, by specifying among prohibited medicaments the large doses of tartar emetic formerly so much prescribed, which place the patient in a state of perilous prostration.

Positive Indications.—These are as follows: to restore the alcoholic stimulant whose sudden suppression has determined the delirium, to combat the agitation and insomnia by narcotics.

Alcohol.—Judicious observers, such as Chomel, Trousseau, Jaccoud, recommend formally the employment of alcoholic stimulants in moderate doses, especially wine, in order not to make too sudden a break in the habits of the patient. It has generally been supposed that the delirium is due to the absence of the ordinary stimulant, for which the brain is now suffering; in this respect the alcoholic patient might be compared to the morphino-maniac, who cannot with impunity be weaned from his morphia. If in fact it is necessary to alcoholize the nervous system, one would have to give (after the fashion of Todd) large quantities, as is the custom in all grave prostrating maladies; if, on the contrary, small doses suffice, (as seems to be the fact), the spirit must act as a sort of attenuated virus, or vaccination, preventive of grave intoxication. It is always the case that in one way or another, alcohol sustains the patient's forces, which are likely to be exhausted by the fever: it is both a dynamic and a refrigerant agency.

Opium.—The second indication is fulfilled by opium, which calms the excitation and favors the critical sleep. Here, as in all the grave diseases, it is necessary to proceed by doses of tolerance; from seven to fifteen grains of opium are indispensable. Small quantities have no effect. But clinicians have protested against enormous doses, rightly affirming that opium in such instances may produce a fatal coma. In fact it is not necessary to prescribe excessive doses, as a moderate exhibition of the narcotic gives the same results, as Ware, Dunglison and Lasègue in particular have shown.

If I may trust to my personal experience, the danger comes from this, that the doses compatible with tolerance, and therapeutical, are continued beyond one or two days; there is thus produced a cumulation, whose effects suddenly manifest themselves at the moment of rally. I have, like Ball, long prescribed opium under the form of subcutaneous injections of mor-

phia; one third of a grain repeated three times a day for two days. This is the best means of avoiding the cumulative action of the drug and the danger of poisoning; if the hypodermic has no effect after that length of time it is best to abandon it.

Digitalis.—In England a physician by the name of Jones was in the habit of prescribing tincture of digitalis in tablespoonful doses to be repeated every four hours; half teaspoonful and teaspoonful doses accomplish nothing, while doses three or four times larger act more on the brain than on the heart and sustain the force and regularity of the latter organ.

Indian Hemp.—Indian hemp may produce sleep immediately: according to Beddoé it has the advantage over opium of not being followed by prostration and stupor. Villard has thus cured seventeen patients out of eighteen. This medicine is prescribed in the dose of twenty drops of the tincture every four hours, or about seven grains of the extract.

I mention only to condemn it, the use of chloroform, which Richardson considers dangerous by the asphyxia which it may produce. I shall only briefly allude to chloral and bromide of potassium, and I protest against expectancy, which nothing justifies.

To sum up, the moderate use of alcoholic stimulants and especially wine, injections of morphia, in the methods and doses indicated, constitute the treatment which seems to me the most rational and which is justified by the experience of practitioners of all epochs.[1]

Treatment of Pneumonia with Pyrexial, Anæmic, or Nervous Delirium.

Hyperthermic Delirium. Antipyretics.—When you have excluded the alcoholic origin of the delirium, you can interpret it only by the hyperthermia, or by the virulence itself of the disease, the malignity, as it used to be called. The indications are then fulfilled by the antipyretics, especially by digitalis, and sulphate of quinine, which support the heart's action, and alcohol in large doses, which restrains heat production, sustains the forces and rapidly calms the delirium.

Antithermics.—If the hyperthermia is very pronounced you will

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[1] There is not consensus of agreement among physicians that delirium tremens is due to a withdrawal of the accustomed stimulus, and facts bear out the assertion that *mania à potu* is a form of alcoholic paralysis, analogous to the *tremblement mercuriel*, the direct consequence of saturation of the nerve-cells of the cerebral cortex with the poison, and of the resulting enfeeblement and perversion of the sensory, perceptive, and motor functions. If this be correct, the leading indication would be to withhold any further exhibition of the intoxicant (*sublata causa tollitur effectus*), and to promote elimination of the poison. This indication, however, must be kept in abeyance for a time by the second, which is the most urgent, for the rapid expenditure of nerve force threatens to wear out the patient. The second indication is to calm the excited brain, and stop the destructive waste going on in the nerve centres till reparation shall be fairly instituted. Thirdly, the system should be supported by suitable nutrients and stimulants. I believe that we have in capsicum the typical stimulant, in chloral the typical hypnotic, and in beef peptones the typical nutrient; the latter should be given in the form of broth or beef tea continuously during the attack, and to the extent of tolerance on the part of the stomach. The judicious use of hypodermic morphia in conjunction with chloral is often necessary, and in hyoscyamine we are likely to have a valuable auxiliary. Digitalis, antimony, crude opium and Indian hemp should be mentioned only to be condemned. —Trans.

obtain refrigeration much more readily by salicylate of soda, in quantities of from a drachm and a half to two drachms a day; but I have seen this medicine cause delirium in typhoid fever, and on this account I should hesitate before resorting to it in cases such as are under consideration.

Prolonged Warm Baths.—It is in these conditions solely that one may with advantage attempt the usage of prolonged warm baths, if there be no obstacle in the way of their application. Possibly it may be worth the while even to try cold lavements, which contribute towards reduction of the temperature and sedation of the nervous system. Local applications, warm or cold, seem to me to be useless, and perhaps dangerous.

Narcotics—Chloral, Opium.—Of these different sedatives the most efficacious is chloral, which is well borne in these delirious pneumonias in the dose of 5 or 6 grammes (75 to 90 grains) in the twenty-four hours; as for opium I have never tried it, and should fear its effect.

Cardiac Medicines—Coffee, Tea.—The latter mentioned beverages are almost always useful. They contain caffein, a valuable heart tonic. Caffein in substance may be given, and the benzoate of caffein, which is soluble, should be tried in grave cases in pretty large doses; 30 to 45 grains a day.

To sum up, after the antipyretics, I prescribe caffein, whose utility is, however, as yet insufficiently established; at the same time baths at 86° F. or cold lavements.

Anæmic Delirium.—This kind of delirium yields readily to generous alimentation and plenty of good wine; all medication seems to me to be useless.

Nervous Delirium—Musk.—The delirium called *nervous*, which cannot be attributed either to hyperthermia, or to *malignity*, or anæmia, has been uniformly regarded as tributary to antispasmodic medication; musk has been the great remedy for this kind of delirium, and was employed by Récamier and vaunted by Trousseau, who formally stipulated the dose of fifteen grains as the sole condition of its efficacy. With this dose one is sure to provoke nausea, vomiting, headache and a sort of intoxication. In moderate doses, no effect from the musk is perceived, unless it be the disagreeableness of its penetrating odor, in other words its excitant action on the olfactory nerves; whether this can give rise to any reflex consequences beneficial to any organ is a matter of grave doubt. It is certain that with us it has become a habit to give musk as a last resort in states of great nervous prostration, and as these states are very often fatal, the very mention or smell of this medicament brings up to most patients very unpleasant associations.

Bromide of Potassium.—Bromide of potassium finds here its use; in quantities of two drachms a day it may render excellent service; the sedative action of this drug in nervous troubles properly so called is no longer under dispute.

Treatment of Pseudo-typhoid Pneumonia.

From the point of view of treatment it seems difficult to dissociate two diseases which, although entirely distinct by their origin, present traits of resemblance by their symptoms; I refer to true typhus with pulmonary determinations, and to those pneumonias called typhoid, sometimes ataxo-adynamic, asthenic, or pseudo-typhoid—the latter being the better term. The former is true typhus (typhoid fever) with pneumonic aspect, and pul-

monary symptoms predominant; it is an interverted typhus, while the latter is pneumonia which has taken the mask of typhoid fever. (See pages 112 and 114.)

Pseudo-typhoid Pneumonia.—This form reveals itself by cephalalgia, general malaise, loss of strength, sometimes epistaxis and even diarrhœa. You are tempted to regard it as a case of typhoid fever, but from the very onset you find fine crepitation, or sub-crepitant râles with the dullness of unilateral or double pneumonia, sometimes central at first, but even then evident by the rusty expectoration. Then supervenes a violent delirium or sub-delirium, with startings of the tendons (subsultus tendinum)—this is the ataxic form—or a hebetude well marked on the countenance, with extreme prostration of the forces, which no longer permits the patient to sit up, dryness of the mucous membrane of the tongue, which is covered with a sooty fur; tympanism of the abdomen, and fetid stools. The picture is complete; there are not even wanting the signs of microbic infection, to wit, the swelling of the spleen and albuminuria.

Natural Resolution.—Now all this frightful syndrome of symptoms may disappear as by magic, spontaneously, or under the influence of divers means of treatment, and the malady then pursues without interruption its regular course.

Indications.—When this form of pseudo-typhoid pneumonia claims the aid of the physician, his first care will be to ascertain the cause of these ataxo-adynamic phenomena. Are they the result of febrile excess? then antipyretics are indicated, especially quinine. If, on the other hand, the temperature is moderate and the adynamic condition has no other possible interpretation than the baneful action of the infectious principle itself on the organic nervous system; if the malady presents, as the ancients expressed it, the characters of malignity, new indications impose themselves; the organism must be by every possible means protected against the parasite which invades it in its entirety, annihilating its forces. Stimulants seem called for and excitants of every kind to arouse the depressed vital powers, but unfortunately the potency of none of these medicines is equal to their ancient repute. The various preparations of ammonia, camphor, hypodermic injections of ether, etc., (anti-spasmodics generally) are commonly resorted to, besides cutaneous revulsion, under all its forms. This, at least, is the traditional treatment; in which, however, I have no faith whatever, believing it to be inefficacious except in the state of collapse. It will be worth the while to consider somewhat at length in the following section these over-vaunted medicaments.

Treatment of the Pseudo-typhoid Pneumonias by the Excitants (Ammonia, Camphor, Etc.).

Treatment by Ammoniacal Preparations. Chemical Properties, Absorption, Elimination.—The preparations of ammonia employed by the physicians of the last century comprise the following substances:

Liquor Ammoniæ.—This is the officinal solution of ammonia, containing (like the similar U. S. P. preparation) 10 per cent. of ammonia. The dose is from 5 to 15 drops largely diluted with water or mucilage. I ordinarily prescribe it after an old formula known under the name of "Anisated Spirits of Ammonia," of which the formula is as follows:

Take of Oil of Anise	1 part,
Alcohol	24 parts,
Water of Ammonia	5 parts.

Dissolve the oil in the alcohol and mix with the water of ammonia. Dose.—Ten drops several times a day.

Carbonate of Ammonium.—This salt, which has an ancient reputation as a diffusible stimulant, is given in doses of five grains in water or mucilage.

Acetate of Ammonium.—This salt is used only in the preparation known as the Solution of Acetate of Ammonium, or Spirit of Mindererus, which contains 15 per cent. of acetate. It is a limpid and colorless liquid without smell, and with a saline taste. The dose is from a teaspoonful to two tablespoonfuls.

Chloride of Ammonium.—The chloride is much in use as an expectorant in bronchitis and its utility, will be discussed in another chapter.

Absorption of Ammoniacal Preparations.—Ammonia when introduced into the stomach is in part neutralized by the gastric juice, and in part absorbed; the salts penetrate the blood in larger quantity.

Elimination by the Lungs.—Ammonia and its salts are constantly eliminated by the lungs, owing to their volatility, although incompletely eliminated. As, however, they undergo decomposition and form new compounds in the blood, it results that this liquid does not give up ammonia in its purity, and that this gas is never found in the expired air. This is what results from the experiments and researches of Böhn and Lange, and of Scheffer;[1] one hardly finds even traces of carbonate of ammonia in the air; ammonia is also lacking in the sweat.

Elimination by the Urine, after Transformation into Urea.—What proves the transformation of the ammoniacal compounds in the blood is that they are eliminated by the urine under the form of urea; the chloride, which, according to Walter, is an exception, also furnishes urea. In injecting into the veins of a dog weighing forty pounds, chloride or nitrate of ammonium, Salkowski has constantly recovered it in part in the urine under the form of urea; the elimination of this excrementitious principle mounted from 5.61 gr. to 9.75 gr. Dreschel has demonstrated with certainty this curious kind of synthesis.[2]

Physiological Effects on the Nervous System, Heart, etc. Experiments.—When you inject dilute water of ammonia or solution of carbonate of ammonia into the veins or under the skin of the higher animals, you observe, by reason of the excitation of the medulla oblongata and of the spinal cord, a precipitated and decidedly dyspnœic respiration, which seems to prove the excitation of the respiratory centres. Then there ensues a series of general tetaniform convulsions which in the hare may end in a mortal tetanus.[3] In the dog it requires a much larger dose to cause death, and when the poison is introduced into the stomach even in the dose of from two to five drachms a day, no very marked troubles of the nervous system are produced.

Vascular Pressure Augmented.—The dominant fact in all these experiments is the augmentation of vascular pressure. Whether the heart be accelerated or not, this excess of pressure is constant. Funke and Deanah explain it by the action of ammonia on the vaso-motor centre. When the pressure has attained its maximum, it ends in six or eight hours by falling to its former level.

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[1] Harnak, Lehrbuch der Arzneimittellehre, 1883.
[2] Arch. physiol., 1880. [3] Funke and Deanah.

Physiological Effects in Man. Therapeutical Effects.—As to the effects of ammonia in the human subject, in whatever doses, whether respired or ingested, we are entirely in the dark. Has any one ever observed under elevated but not toxic doses that excitation of the respiratory centres which seems so dangerous, and that arousing of the vaso-motor centres which in producing a strong intra-vascular pressure may serve to raise the force of the heart? There are no positive observations of the kind whatever. It is nevertheless for this object that ammonia in its various preparations is prescribed, and it may subserve some such physiological purposes as are above indicated in all the irregular pneumonias and particularly in the pseudo-typhoid forms. Outside of these indications I do not see any use in ammoniacal preparations, although Patton has instituted a regular system of treatment by carbonate of ammonia.

Camphor. General Considerations.—Like all its congeners of the ill understood and possibly fabulous class of anti-spasmodics, camphor, which had a great rôle in the medical practice of the last century, has been considered as sometimes an excitant (if so it cannot be a calmative), sometimes as the check regulator of spasms (if this be the case it can hardly be an excitant). Finally as it kills mites and preserves garments from the moths, it has the universal reputation of being a parasiticide, and it is on this ground, probably, that it figures so largely in the history of putrid or malignant diseases.

Chemical Properties and Preparations.—Camphor, almost insoluble in water, is given in the dose of one to five and even ten grains in pills or in emulsion, the latter containing fifteen grains to be distributed through the day; it is in this manner that Jurgensen prescribes it. By the side of camphor should be mentioned the ethereal oils, menthol, etc., which are not of easier administration, and valerian, which contains some of the camphor principles.

Physiological Effects.—On cold-blooded animals camphor produces an effect similar to wourara, and this sort of curarization prevents the development of convulsions. Moreover the *cardiac* muscle, whatever may be the mode of administration, receives a stimulus which is capable of rallying the heart poisoned by muscarine, or paralyzed in its motor-ganglia, or enfeebled in its muscular excitability. (Schmiedeberg.) This is a very interesting property.

In the higher animals and in man you sometimes witness the supervention of violent and repeated epileptiform convulsions, which dominate the scene of poisoning in such a manner as to overshadow the respiratory troubles and the acceleration of the pulse arising from the excitation of the medullary centres of respiration and of the heart.

In animals which have been previously curarized, we may note also an excitation of the vaso-motor centres which manifests itself by an augmentation of the blood pressure.

Certain cerebral manifestations sometimes precede the convulsions. In the dog a sort of continuous propulsive movement is observed. In man there have been noted, but only after large doses of from forty to sixty grains, attacks of vertigo, cephalalgia, and mental aberration, then after a brief acceleration, a notable slowing of the pulse, and finally loss of consciousness with convulsive phenomena. (Schmiedeberg.)

Therapeutic Effects.—It is by reason of its excitation of the respiratory and vascular centres that camphor seems to benefit in the collapses which follow adynamic fevers; the respiration becomes more energetic,

the heart's activity is restored with that of the vaso-motor centres, which lose their excitability in states of general and circulatory prostration. It is, then, in augmenting the pressure, and in accelerating the movements of the heart that camphor exercises its curative properties, which are unfortunately limited by its volatility, its insolubility in water, and consequently the difficulties attending its employment.

Effects of Camphor and Odorous Substances in General.—It is precisely this volatility, like that of ammonia, which is utilized in the treatment of syncopal states. These substances in vaporizing act by their penetrating odor on the organs of olfaction, *i.e.*, on the first and especially the fifth nerves; there results a centripetal excitation of the inhibitory cardiac nerves—the vagi—which manifests itself in a slowing of the pulse, and at the same time an increased intra-vascular pressure. All these phenomena have been well studied by Hering and Kratschmer, by Kirshaber, by Marey, and François Frank. A final very important effect is produced, namely, a reflex excitation of the bulb itself, and consequently an arousing of the respiratory function in states of syncope and asphyxia.

Injections of Ether.—We have in hypodermic injections of ether a powerful means for combating states of collapse; this mode of treatment is not, however, to be persisted in; it is not even applicable to the ataxo-adynamic forms.

Resume.—We have learned practically nothing respecting the treatment of pseudo-typhoid states. We have acquired therapeutic notions of great utility concerning collapsus, which is so common an event in the course of these asthenic pneumonias.

Treatment of Bilious Pneumonia. History of Emeto-cathartics.

The history of bilious pneumonia and that of emeto-cathartics go together. We are led back to the first days of the humoral pathology, entertained by the immediate successors of Hippocrates who abandoned the naturist idea; the blood, the bile, the atrabile, and the phlegm already played an important part in the genesis of diseases. Galen was a marvelous discerner of general diseases, local affections, and of primarily local diseases which become generalized; he greatly dreaded such generalization. When an acute malady threatens to invade the entire body, you must anticipate and prevent this, he said, by a free bleeding, except in the case of a drunkard (which showed a rare judgment). After the blood-letting he prescribed emetics to expel the bile, and purgatives to remove phlegm. This fantastic iatro-chymiatry dominated the minds of medical men during long ages, and acquired with Sylvius (or Dubois), a more definite shape under the name of fermentation. In the seventeenth century, Sydenham protested against the notions of the chemists, to combat which he invented the theory (which has long been so popular) of medical constitutions, called inflammatory, bilious or adynamic, influencing all the prevalent diseases and the treatments appropriate to these constitutions. But it happened that *after*, as well as *before*, the renovation of these constitutions which dominated the entire pathological scene, physicians never failed to resort to bleeding and to emeto-cathartics; no acute disease escaped the prescription, small-pox as well as measles, and cholera as well as pneumonia were treated by these violent procedures.[1]

[1] In justice to Sydenham it should be stated that he taught and practised a more rational treatment of febrile diseases.—Trans.

The rule was absolute; it was necessary to begin by evacuants, no matter what "medical constitution" might be supposed prevalent. The profession continued to march in accordance with these marvelous principles till, at the end of the eighteenth century, there was no longer anything in fashion but the "bilious constitution." Stoll was chiefly instrumental in giving ascendancy to this notion; he at one time witnessed an epidemic of "bilious pneumonia," of which he reports three cases, and the name henceforth had a permanent place in medical literature. We now know that these bilious pneumonias are pneumonias with gastric catarrh predominant—what we call at the present day the gastric, or saburral, or gastro-bilious states; in most of these cases vomiting does not bring up any bile; this, however, was not then known, or if known, did not change the situation; it was always bilious pneumonia with Stoll, which, nevertheless, he perfectly distinguished from pneumonia with icterus, of which he relates four examples.

This, then, was the famous bilious pneumonia, grave or benign; it was this which imposed the obligation to give emetics, and which still possesses the minds of Stoll's modern successors. Regarding the bile as cause or as effect, to this they directed their therapeutics, and prescribed emetics or cholagogues; if the theory is wrong the practice is equally to be condemned.

Pneumonia with Vomiting is not Bilious Pneumonia.—An error which has been often committed is to reckon the vomiting of pneumonia among the symptoms of the state called bilious or gastric; now we know that in the case of children especially, pneumonia is almost constantly ushered in by vomiting. With regard to the acute febrile diseases which begin by vomiting, it seems to me that we might establish a hierarchy, and formulate a pathological law:—pneumonia, scarlet fever, small-pox commence by vomiting much more frequently than broncho-pneumonia, measles, typhoid fever; this is the rule; the pathological law which I propose is this: When the fever begins with great intensity and suddenness, it provokes in the functions of the vagus system (medullary centre, or peripheral terminations), an irritation much more marked than when the pyrexia rises gradually, as in the instance of typhoid fever, where the thermometrical ascent is by successive halts. In these cases the elevated temperature does not oppress the nervous system as it does in the former class of cases where the initial temperature is high. The phenomenon is, moreover, more marked in children, whose excito-motor power is always more pronounced than in the adult; the vomiting is, then, a reflex nervous manifestation of sudden hyperthermic origin, and has no relation to the state of the biliary secretion.

Pneumonia with Jaundice is but rarely Bilious Pneumonia.—The icterus may be benignant and oppose no interruption to the regular march of the pneumonia; it is the result of blood stasis in the liver, or else it may depend on the extension of the intestinal catarrh to the common bile duct, when it becomes a true bilious icteric pneumonia; it may, however, be of alcoholic nature, or, lastly, of infectious origin, constituting the grave bilious pneumonias which have been late years described.

Treatment of Bilious Pneumonia by Emetics and Emeto-cathartics.

All the common means and all the medicaments which cause vomiting are not necessarily emetics. A great number of pharmaceutical substances

excite the antiperistaltic functions of the stomach, either directly or by the intermediation of the terminal pneumogastric filaments of the gastric mucous membrane.

When arsenic is taken in toxic doses it acts on the mucous membrane, causing vomiting, or if it is absorbed it is eliminated by the digestive passages, and determines the effects of an emeto-cathartic. When digitalis is ingested, it provokes, whether at the onset, or after a prolonged or excessive usage, vomitings which are nervous in their character; these are not emetics properly speaking; the name is more correctly applied to medicaments whose principal effect is to provoke vomiting without causing symptoms of poisoning either before or after their ingestion. We may, then, define EMETICS as certain pharmaco-dynamic substances which act primarily on the terminal extremities of the gastric vagus nerve, in such a way that the impression felt is transmitted to the bulbar centre and determines a convulsive effort of the auxiliary abdominal muscles of expiration, which on contracting violently, expel the contents of the stomach.

Under this title we now employ only tartar emetic and ipecacuanha, which owes its principal action to emetia. A third substance, which possesses in a much higher degree the emetic power, is *apomorphia*, which is a derivative of opium, but which differs sensibly from the two first, because it produces at the same time a predominant action on the heart and the striped muscles. We need not here touch upon other emetics, such as violine, obtained from the violet, or sulphate of copper, to which has been attributed valuable properties in the treatment of croup.

We shall now concern ourselves only with tartar emetic, whose therapeutic applications we shall have to investigate, its physiological effects having been already discussed at length. We shall next study ipecacuanha, then apomorphia in a special paragraph.

Effects of Emetics and Emeto-cathartics.—Multiple effects have been attributed to emeto-cathartics, which have always and everywhere been considered advantageous; such as the evacuation of the gastro-intestinal contents, including the bile; a special irritation of the mucous membrane and by this very effect a derivation to the digestive organs; a mechanical influence on the secretions of the respiratory organs; a depletive action on the circulation; diaphoresis, and the sedation of the central nervous system.

Evacuant Effects.—Tartar emetic alone or with the addition of a purgative salt does not effect evacuations, as is generally believed, by acting as an irritant to the stomach and intestines; this is the secondary result of the emetic absorbed by the blood, and eliminated by the mucous membranes. It is at the moment of its elimination that the emetic produces on the vagi nerves and their terminal branches in the stomach, a centripetal impression which gains the medulla oblongata and is reflected on the expiratory nerves and muscles. Whether the stomach contain mucus in appreciable quantity or those products called saburral, which consist in a layer of mucus mingled with epithelium, such as one sees so often on the mucous membrane of the tongue, these secretions are evacuated, with or without alimentary matters. If bile is mingled with the vomited matters, it is not because the emetic has provoked a hyper-secretion of bile or a more marked excretion of this fluid secreted in excess; it is simply a mechanical act, which by compression of the abdominal muscles, causes a reflux of the bile into the upper part of the duodenum and from thence into the stomach.

The most remarkable consequence of artificial vomiting is the cessation of the nausea and want of appetite and the clearing of the coating from the tongue; the patients are certainly relieved, but this is all. The emetic has not expelled the offending body, that is to say, the bile which is not in excess, nor the saburral matter which does not exist; it has only removed the mucus which in fevers always embarrasses the stomach and causes a sort of temporary mucous dyspepsia.

Irritant and Derivative Effects on the Mucous Membrane of the Stomach.—All that has been imagined about the direct local irritant and revulsive effect of tartar emetic on the stomach and intestines is simply so much romancing; it does not directly and immediately act upon these organs, and we know that it does not cause vomiting till it undergoes elimination by the mucosa. The antimonial not only does not irritate the stomach, but it clears it of the mucus which encumbers it, and thus favors the secretion and action of the gastric juice and permits the digestion of simple liquid nutrients.

Mechanical Effects on the Air Passages.—The emetic acts mechanically on the laryngo-bronchial tubes to eliminate their contents, just as it acts mechanically on the digestive passages; this is a fact acquired to science, and which finds especial verification in the bronchial catarrhs treated by ipecac as will be shown in another chapter.

Depletive Effects on the Circulation.—The view having been abandoned that the antimonial produces a derivative effect from the nervous centres to the digestive passages, freeing the former of the irritation under which they are laboring, by producing in the stomach and intestines a substitutive irritation, it has been alleged that emeto-cathartics have a depletory action on the greater and lesser circulation by the drain which they occasion; the blood losing thus its surplus and its serum. This is still an hypothesis which could not be realized unless the mass of evacuated liquids should be sufficiently abundant and the evacuation sufficiently repeated to constitute a sort of serous bleeding. Now we know that emetics eliminate only the gastric juice, food and mucus in the stomach, and that purgatives carry off, besides fæcal matters, only the intestinal, biliary and pancreatic juices which are naturally in the intestines; in this way the blood is not directly impoverished, but the patient is impoverished of his digestive juices.

To sum up, we see that the entire scaffolding of emeto-cathartic theories crumbles before the rigid examination of the facts, and there is left to justify the usage of these medicaments, only the benefit derived from their mechanical effect.

Treatment of Exaggerated and Complicated Pneumonias.

The most regular pneumonia may be aggravated by certain local symptoms, such as the pain and dyspnœa, be attended by certain lesions of the circulation, such as pulmonary œdema, or be complicated with concomitant affections, such as pleurisy (which is seen in the proportion of 11 per cent.), bronchitis, alterations of the membranes of the heart, and especially myocarditis with or without degeneration of the heart muscle. With regard to these complications the reader is referred to the respective chapters which treat of pleuro-pneumonia, bronchitis, and the affections of the heart. The predominance of the pain, dyspnœa and œdema may be such as to demand the interference of the physician, who ought not, how-

ever, to lose sight of the normal march of the disease, and the gradual abatement of the distress which attends such natural evolution.

Thoracic Pain.—The pain in the side augments generally till the third day and then abates spontaneously; it may manifest itself with great intensity (whatever may be said to the contrary) without the least sign of dry pleurisy, and in pneumonias of the apex as well as in those which occupy the middle or lower lobes. As this pain is sometimes so severe as seriously to hinder the respiratory chest expansion, and thus to augment the dyspnœa, there will be an urgent indication to relieve it, which may be accomplished first and most certainly by *hypodermic morphia;* this not only alleviates pain but it facilitates respiration. The *vesicatory* is a French method of treatment, which had better, however, be reserved for the dyspnœa, where it can render an effective service. *Applications of cold compresses* are vaunted in Germany; they certainly may be a means of provoking a pleurisy which is lacking. *Wet cups* appear to have received general approbation; it is, however, needless to say that they are contra-indicated in children, old people and debilitated individuals, and they are in fact rarely needed to combat the pain, the subcutaneous morphine injection being every way a preferable means.

Dyspnœa.—The dyspnœa has not always the same source; it depends, either on the pain, the asphyxia, or the hyperthermia. In the first case, the hypodermic injection of morphia will give prompt alleviation. The second case demands a special study. "When asphyxia is imminent," says Jaccoud (who, by the way, has never found this urgent indication), "a large blood-letting is indicated;" we have here pulmonary œdema superadded to the inflammatory lesion. In simple pneumonias, whatever may be their extent, the dyspnœa is rarely dangerous, and can easily be relieved by the most simple means; bleeding is too much of a remedial measure, and leaves its effects in the organism. Wet cups have the inconveniences without the advantages of general blood-letting; *blisters* ought not to be employed except at the termination of the disease, because they augment (it is said) the congestion: the cold bath, which is reputed to give such agreeable sensations to the patient, is likely to cause him an increase of the dyspnœa; in short there is nothing so good, again, as the subcutaneous injection of morphia, to which I habitually resort, and which is, as Huchard has shown, a respiratory medicament of the first order and perfectly applicable to pneumonia. Therapeutists have long ceased to impute congestive properties to morphia; which really opposes congestion.

In fine the thermic dyspnœa, recently studied experimentally by Charles Richet, is a central thermo-dyspnœa which is manifested by an enormous acceleration of the respirations when once the central temperature attains 104° F., and it must not be confounded with the reflex dyspnœa which results from external heat. Antipyretics must be freely used to combat the causes of this dyspnœa.

Pulmonary Œdema by Excess of Activity or Feebleness of the Heart.—This dyspnœa is the sign of an œdema which Niemeyer has called collateral, and which he attributes to an artificial fluxion which is produced in the branches of the pulmonary artery remaining permeable; the inordinate activity of the heart causes also an extraordinary afflux of blood to the alveoli and a serous transudation in the interstitial tissue around them. This œdema, which Jurgensen claims never to have seen but twice, and which never had an existence except in the imagination of

Niemeyer, would naturally demand blood-letting as the most ready and effectual remedy.[1]

Œdema by weakness of the heart, œdema by stasis of the blood in the pulmonary vessels—this is what one frequently observes; it is an obvious inference that the left heart is more readily and more thoroughly fatigued than the right heart. When the left ventricle can no longer propel the blood, there results, despite the efforts of the right heart, a repletion of the lesser circulation, and from this circumstance an œdema by stasis. (Cohnheim.)

Treatment.—It is not blood-letting that can remove this stasis or this cyanotic asphyxia; this grave condition necessitates the employment of the most energetic excitants, such as injections of ether, the internal use of wine, of camphor, of coffee, the external application of the most promptly acting revulsives. One may thus succeed in arresting this grave incident—this insufficience, this collapse of the heart, which in every respect resembles collapse of the general forces, and demands the same treatment.

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[1] Niemeyer, Pract. Med. (Appleton's Ed.) vol. i., p. 168, enumerates among the cases of dyspnœa, "collateral œdema in the inflamed part of the lung, which causes swelling of the vesicular walls and decrease of their capacity." Under the head of "treatment" he makes this collateral œdema an indication for the prompt letting of blood—"When collateral œdema in the portions of the lung unaffected by pneumonia is causing danger to life, the pressure of the blood is reduced by bleeding; and by prevention of further transudation of serum into the vesicles, insufficience of the lung, and carbonic acid poisoning are averted. Whenever the great frequence of respiration in the commencement of pneumonia cannot be traced to fever, pain, and extent of this pneumonic process alone, *as soon as a serous frothy expectoration appears, together with a respiration of forty or fifty breaths a minute*, and when the rattle in the chest does not cease for a while after the patient has coughed, we ought at once to practise a copious venesection, in order to reduce the mass of blood and to moderate the collateral pressure."—Trans.

CHAPTER XXXI.

TREATMENT OF THE SPECIFIC PNEUMONIAS.

Under this name are comprehended all the pneumonias which recognize a specific origin. In malarial pneumonia the parasite which produces it comes from the soil and diffuses itself in the air under the name of marsh miasm, as is to-day thoroughly demonstrated. As for influenza, it takes its origin in the atmosphere, and diffuses itself so as to constitute a disease essentially epidemic. Typhoid fever exists only in virtue of a parasite of which the fæcal matters and the water are the vehicle. And even rheumatism is suspected of also deriving its source in a microphyte.

Malarial or Intermittent Pneumonias.

Nature of Malarial or Intermittent Pneumonias.—From an etiological point of view, which is the sole point which interests us, marsh pneumonia is not under dispute; the contention is only concerning the character of intermittence which it habitually takes on, like the intermittent fevers which are from the same cause. The doubts which exist pertain to the nature of the disease, since all the phenomena, signs and lesions of this periodical marsh pneumonia do not disappear completely with the fever paroxysm which marks its onset and characterizes its march. The intermittence is not absolute, as it is in the case of the comatose, deliriant, algid, larvated, dysenteric forms, whose distinctive symptoms (coma, delirium, neuralgia, etc.) subside in the intervals of the paroxysms.

We have here profound lesions of the lung, engorgement, hepatization, in a word, fibrinous pneumonia in its various stages, or possibly lobular or even lobar broncho-pneumonia. In the interval of the paroxysm (period of apyrexia) you still notice, it is true, the sanguinolent expectoration, crepitant râles and even the bronchial souffle; the disease is, however, as much as ever under the dependence of the malarial virus, and the curative agent, quinine, will have its usual anti-malarial effect.

In fact, whether it take on the tertian or quotidian type, whether it be ushered in or not by the ordinary ague fits, the pneumonic fever is so completely subject to the malarial element on the one hand, and to the fever on the other, that it commences, like any pernicious fever, by a profound initial chill, with extreme prostration and restlessness, after which there is a remarkable lull, as when the regular ague fit terminates —the pain in the side, the dyspnœa cease; if, however, the crepitant râle, the souffle, the bloody expectoration persist, it is owing to the fact that the lesion is in delitescence without having undergone resolution, and that functional troubles remain which menace the life of the patient. If a second paroxysm appear, the entire series of symptoms is reproduced, to again subside at the decline of this second attack.

We have here something besides ordinary pneumonia in an ague patient, as Rayer maintained, who judged the disease only by its anatomical side. It is the malarial infection seizing upon and localizing itself in the lungs; this is the decision of Torti, who knew only the functional troubles, of Laennec, Chomel and Grisolle, who studied it with the help of the stethoscope, while Catteloup, Armaingaud and Purjoz followed with thermometer in hand, the dramatic march of the malady.[1] [Griesinger, as cited by Hertz,[2] compares the filling up of the lung that takes place under the paroxysms of malarial fever to the enlargements of the spleen, which at first recede during the intervals of the fever, but afterwards remain constant. If timely recourse to quinine is neglected these cases are apt to end fatally at about the fourth or sixth paroxysm.]

Sulphate of Quinine in Intermittent Perniciosa Pneumonica.—The result of the quinine treatment is a forcible protestation against the theory of "complications," or "fortuitous coincidences." If you were to be so unfortunate as to mistake in your diagnosis, and were to treat your case as one of simple complicated or uncomplicated frank pneumonia, and were to prescribe blood-letting with or without small doses of quinine, you would infallibly lose your patient. It is important to give the quinine in massive, anti-parasitic doses, for it is a micro-organism invading and destroying the patient through the lungs against which you have to contend; you must then strike a heavy blow by a full large dose, followed by smaller doses, according to Torti's method, who was the first carefully to study pernicious intermittent pneumonia. To-day we prescribe a gramme (15 grains) to be taken at once, and a half gramme or a gramme to be taken several hours afterwards. Another precaution more indispensable still is this: if the patient is in the paroxysm, make him take the first dose at the decline of the paroxysm, during the sweating stage; the circulation being re-established, tolerance is effected immediately. If you arrive at your patient's residence before the second paroxysm, although not more than an hour before, prescribe the large dose immediately, and for a still stronger reason if there be still five or six hours of respite to wait before the next attack. Do not forget, however, that the second attack is often sub-intrant. Six hours certainly suffice for the medicament to stop the paroxysm; the period of one or two hours is enough to moderate the threatening paroxysm, and take from it its perniciousness, and especially to prevent the third paroxysm, which might be fatal. The second dose will come to support the effect of the first, and bring security to the mind of the physician. For still greater safety it will be desirable to continue the same doses for two or three days.

I have seen a case of this kind in Paris in a man thirty years of age who had never had intermittent fever; another case in an officer who after a long sojourn in Africa had returned to France, where he had been stationed three years at the Fort Chatillon; I was called before the second paroxysm. Both patients got well without having to go through with the third attack.

Treatment of Pneumo-typhus.

Pneumo-typhus is a species quite distinct from pneumonia of the frank fibrinous kind. Whether this parasitic affection be regular or marked by that train of symptoms which constitute ataxo-adynamia or pseudo-

[1] Arch. Klin., 1884. [2] Ziemssen's Cyclopædia, Wood's Ed., vol. ii. page 612.

typhism,[1] it little matters; the virulent principle which produces both differs altogether, and the course of the affections is different.

It is possible that the pneumonia may mark the terminal period of a case of typhoid fever and be only a complication, or that the two virulent affections may march side by side without any mutual influence being exerted on each other, or finally, that the pneumonia may be the only manifestation from the onset, concentrating in itself (so to speak), the entire typhic process. In the first case the treatment would be that of a typhous complication; in the second it is the treatment of two diseases that is demanded—diseases which are united, and yet separate. The chief difficulty is in reference to pneumo-typhus, which is not easily diagnosticated from the ataxo-adynamic pneumonias of microbe origin, and which is still more difficult of treatment in accordance with its special origin. What we have to do, in reality, is to treat typhoid fever with pulmonary, instead of intestinal localization. The indications, in default of specific medication, will be fulfilled by the antipyretic and supporting treatment; by quinine and alcohol. In the discussion before the Academy of Medicine in January, 1883, this was my conclusion, and the less absolute conclusion of Jaccoud. The refrigerant (cold bath) treatment, which was then under dispute with reference to typhoid fever in general, is of still more doubtful expediency in pneumo-typhus.

Treatment of Rheumatismal Pneumonias.

Among the microbe diseases badly defined but certain in their origin, we must henceforth count rheumatism. According to the view which we defend, rheumatismal pneumonia comprehends neither the pneumonia *à frigore* which we have described, nor the infectious pneumonia which may accidentally develop as a complication of acute articular rheumatism, for the fact that a patient may have rheumatic fever with or without pericardial or endocardial complication does not by any means exempt him from all other infectious diseases. True rheumatismal pneumonia does not exist except under the dominance of articular rheumatism in a patient suffering from the disease; under such circumstances the pneumonia is specific, but after a different manner from ordinary pneumonia; it is a distinct species like pneumo-typhus, and to avoid confusion we may call it *pneumo-rheumatism.*

Now pneumo-rheumatism manifests itself in different ways: it is in the course or even in the decline of general articular rheumatism that it attacks the lung, sometimes at the same time the pleura and pericardium. In these cases the pneumo-rheumatism does not influence the march of the articular rheumatism, and is not influenced by it, but constitutes an aggravation. At other times, on the contrary, it has the appearance of alternating with the arthritides, and of only manifesting itself simultaneously with subsidence of the joint swellings, as if the poison had left the joints to bring all its force to bear upon the more important vital organs. This is what is called metastasis. But in these cases it is vain to try to recall the arthritic determinations or to cause derivation of the disease to the external surface by the most energetic revulsives. You can no more in this way cause an abatement of the pulmonary phlegmasia, than you can by counter-irritants, or "derivatives" cure rheumatic pericarditis or endocarditis, as Bouillaud proved beyond a cavil, in opposition to Chomel

[1] See page 144.

and the believers in metastasis. What we really observe in these cases is an extension of the disease, and not a mere displacement, or alternation. You have to do with the rheumatic virus itself, which, after having remained for a time in its place of predilection, has diffused itself and attacked the vital organs, and the principle of metastasis no more finds its application here than it does in gout, which physicians the world over fear to dislodge from the joints.

A third mode of development of internal rheumatism is still more a subject of scepticism and suspicion; I refer to its manifestations in the lungs or heart after complete cure of the joint affection, whether by the old quinine treatment or by salicylate of soda. Have we not heard it said that it will not do to cure articular rheumatism too early and too quickly for fear of seeing the disease invade the interior of the organism? But it is precisely the opposite which takes place; all observers in all countries are to-day agreed, not only to absolve the salicylate medication from this imputation, but even to attribute to it the marvelous property of arresting the disease at its fountain-head. Instead of 80 per cent. of pericardial and endocardial complications, you now have but 5 per cent., as statistics prove.[1]

The real difficulty is not there; it concerns the treatment of pneumonias which continue their work of invasion and extension despite the salicylate, or when it has been administered at too late a date. These pneumonias are rarely benign and fugacious, whatever Sturges may have said. I have always seen them take on a character of exceptional gravity. How is it best to combat them; by still giving the salicylate? Without doubt, if, as in articular rheumatism, it calms the pain and lowers the temperature, ordinarily excessive, which marks these generalized rheumatisms—*totius substantiæ;* this is, however, all the benefit that you can realize from this medicament in many cases. While the articular fluxion soon subsides, and completely, for the very reason that it is only an imperfect kind of inflammation with a local microbe, rheumatismal pneumonia lamentably resists the employ of the salicyate and often ends in death; it is a special pneumonic infection. One is, then, obliged to have recourse to antithermic means, and especially alcohol: it is our only resort.

Variolic, Diphtheritic Pneumonias.—These are grave forms of pneumonia, often complicated with fatty degeneration of the heart and endocarditis. They will be considered in another volume.

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[1] Langer, Kl. Blätt., 1884.

CHAPTER XXXII.

TREATMENT OF INDIVIDUAL FORMS OF PNEUMONIA.

The individual conditions of age and previous health exercise a great influence on the indications of practice. Children, old men, persons affected with chronic diseases, such as Bright's disease, diabetes, gout, heart affections arising from these and other causes—who are attacked with pneumonia, claim care and attention altogether different from the ordinary means of treatment.

Infectious Pneumonia in Children.

Regular Pneumonia.—In children of from two to fifteen years of age, regular pneumonia always ends in recovery. This is my experience, which is in harmony with that of our best observers, Legendre, Rilliet and Barthez, Bergeron, Cadet de Gassicourt, Picot and Despine. If this is the rule, there is another law no less rigorous, namely, that in the child, as in the adult, there is a pneumonic cycle which nothing can interrupt or disturb—unless it be a bad kind of treatment—till the day of defervescence, which arrives often a little sooner than in the case of the adult; the sixth, instead of the seventh day. Why, then, speak of therapeutic interference? This is my reply: the parents never share the convictions of the physician; they demand his help, even if it be for nothing but to prescribe broth and milk; hence it is necessary for him in order to allay their impatience to make certain harmless prescriptions; this is demanded for a still stronger reason when there is an excess of pyrexia. In the latter event you may order from one to two fluid ounces a day of cognac to be taken in the drinks or nourishment of the little patient (as milk punch for instance), or doses of a teaspoonful, or a fraction of a teaspoonful, of infusion of digitalis, or even a mucilaginous lavement containing a couple of grains of quinine; this is especially indicated if the morning remissions of temperature are well marked.

Pneumonia of Infants during Lactation and Dentition.—In infants under two years who suck, or who have been weaned, or who are going through the first dentition, pulmonary inflammation has a slower and more uneven march; if, however, it be true pneumonia, recovery takes place as at more advanced periods of life. The exceptional gravity in very young infants, alluded to by several good observers belongs without doubt, as Cadet de Gassicourt has shown, to a disease of different nature; in fact we oftener meet in young infants with pseudo-lobar broncho-pneumonia which does not follow the regular course or the benign march of true pneumonia. (See Chap. XXXV., p. 237.)

Cerebral Pneumonia.—In the adult, cerebral pneumonia manifests itself by delirium; in the child, by coma or by pseudo-meningitis, or by convulsions.

Pneumonic-coma.—This is the most common form of cerebral pneumonia in the child of from two to ten years old; it is more rare before and after this time of life. The disease commences insidiously by a violent fever and cerebral symptoms, which the medical attendant seldom fails to treat as a congestion or even as meningitis by leeches over the mastoid process and by intestinal derivatives. It suffices, however, in order to avoid this grave mistake, to know that there is no such thing as primary cerebral congestion in the young child, and that meningitis has a quite different course; and especially to remember that the brain may be attacked at the onset of pneumonia. The first duty is to auscultate the chest in order if possible to discover the cause of the cerebral accidents, or in other words, the pneumonia, whether it have its seat at the base, or, as is more frequently the case, at the apex; in the latter event the exploration of the sub-clavicular region and of the supra-spinous fossa will not always give the characteristic signs, that is to say, the blowing breathing and the dullness which alone distinguish the pneumonia of infancy, the crepitant râle being always absent. These signs are better perceived in the axillary region, and it is there that you should search for them. It is the same in the case of central pneumonia, which may occasion some embarrassment in the matter of diagnosis before the inflammation reaches the surface, and even in this event it shows a predilection for the axillary region; it is there that I found it in my first patient, nearly forty years ago. This was a child four years old that had been treated for ten days by Guersant and other eminent physicians without the disease being detected; I was then night interne, and discovered the hidden mischief, and the pneumonia got well like an ordinary case, on the twelfth day.

In patients of this kind be not yourself disturbed, and above all do not worry the child; as soon as the pneumonia is found out you can practise expectancy without fear, as in the simplest cases of frank pneumonia; neither leeches nor derivatives nor musk nor quinine will abridge for a day the henceforth regular cycle of the disease.

Pseudo-meningitis.—I may say as much of the pseudo-meningitic pneumonias, which begin by vomiting, cephalalgia, somnolence alternating with jactitation or even convulsive agitation. Meningitis has not that initial fever which is so excessive in pneumonia, nor those muscular tremblings at the onset. Here also as soon as you have recognized the pneumonia, you may be sure of your prognosis, and you ought to be reserved in your treatment; I do not even allow bromide of potassium, which may debilitate the child.

Pneumonic Convulsions.—Very rare after two years of age, convulsions frequently mark the onset of pneumonia during the time of nursing, of weaning or of dentition. Before the age of two years the excito-motricity of the spinal cord is more developed than at a more advanced age; hence there are convulsive movements from trifling causes, from unconscious as well as from painful irritations. It is difficult in these cases to remain passive; I should not fear to prescribe moderate inhalations of chloroform to arrest the convulsions which may by themselves compromise respiration and life. I have always seen the anæsthetic borne with safety and advantage; afterwards you may await patiently the ordinary evolution of the disease.

Pneumonia of Old Age.

I. Simple Pneumonia.—The pneumonia of advanced life is in respect to fatality, quite the opposite of that of childhood, being as grave as the

latter is benignant. Between the ages of fifty and sixty, the mortality at Vienna is 40 per cent.; between sixty and seventy it is 48 per cent.; beyond this age more than two thirds die. At Paris, the statistics have furnished Grisolle with less formidable figures.

Treatment.—The practice of venesection is happily abandoned. The distinction which was formerly made between old people that were stout and possessed of considerable vitality, and those that were lean and spare, is practically null and void when you come to the treatment of a disease so fatal to the aged; and even when there is an appearance of force we should not forget that if the heart beats with vigor, it is because it contends, with a persistence which soon exhausts it, against senile lesions of the arteries and against the obstacles to the circulation which these lesions bring in their train. Tartar emetic in repeated large doses is also to be ruled out, this medicament being essentially a cardiac depressant, and in large doses it is certainly a paralyzer of the heart, as it is a poison of the muscular system generally. Revulsives do nothing but augment the febrile movement which is always so detrimental to the aged patient.

There are no other indications to fulfil except to sustain the forces of the patient by means of alcohol in large doses, and at the same time to clear the bronchi of the mucus which threatens to obstruct them, and which the senile patient has not always the strength to expectorate, or even dislodge. This object can generally be accomplished by means of ipecac. or tartar emetic in an emetic dose. In the interval of the emetics one may without fear prescribe kermes or James' powder in fractional doses; the debilitation which might result from the use of these medicines is largely compensated by the alcohol, to which one might add digitalis or quinine.

II. Cerebral Pneumonia.—There is between infantile and senile pneumonia one single point of similarity, pertaining to the cerebral complications. Some old patients, says Lépine, fall more or less suddenly into a comatose apoplectiform state with unilateral paresis and hyperthermia; death supervenes in the midst of the coma which the pneumonia has provoked. This sudden and fatal termination causes surprise; the absence of all cerebral lesion, and often the little extent of the pulmonary lesion, make both interpretation and treatment a matter of difficulty.

Pneumonia during Gestation.

Pneumonia occurring during pregnancy demands special precautions from the point of view of treatment. Venesection, recommended by my regretted friend Depaul, and by Peter, has not been adopted in a general way, while doubtless preferable to emetics and especially tartrate of antimony; the latter is an abortifacient, and the same may be said of quinine. Digitalis is quite safe, and under almost any circumstances alcohol is indicated, besides the usual dietary régime of broths, milk, and nourishing gruels.

CHAPTER XXXIII.

TREATMENT OF PNEUMONIA COMPLICATING CHRONIC DISEASES.

Among the conditions which exercise a marked influence on pneumonia, we must reckon previous states of health: I understand by this, anatomical lesions of the heart, lungs, kidneys, chronic diseases such as gout, alcoholism, diabetes, obesity, certain conditions of greater or less gravity and persistence, such as anæmia, over-exhaustion, and physiological depravement.

Cardiac Pneumonia.—It is remarkable that pneumonia is one of the rare acute and infectious diseases that does not alter the myocardium. Autopsies of patients dying from pneumonia do not reveal myocarditis, but observations show during life a diminution of the heart's action, especially of the left heart, which in the adult is more easily exhausted than the right heart. It is this fatigue of the heart, especially likely to occur in individuals badly nourished, or in a state of relative inanition, which constitutes the danger of these pneumonias.

For a stronger reason, when the heart has an incessant struggle to maintain against obstacles in its valves or in the blood-vessels, it is liable to give out; in these cases the compensatory action rapidly fails by reason of previous lesions impeding the general circulation, and the serious hinderance opposed by the pneumonia to the pulmonary circulation. The cardiac patient affected with pneumonia is in perpetual danger, and the peril comes less from the parasitic infection, than from the extent of the pulmonary lesion, which leads directly to asphyxia from the moment that the heart has lost its compensatory power, that is to say, its muscular force, its contractile energy.

Under such circumstances the use of digitalis is imperative, and local derivatives also find their indication. Blood-letting even seems to have some justification as an adjuvant to the unloading of the lungs, but it will not do to forget its bad effects on the muscular fibre.

Pneumonia of the Gouty.—There is no gouty pneumonia, as there is a rheumatismal pneumonia; the gouty patient may have during his paroxysms, or in their interval, an attack of ordinary infectious pneumonia, which may be regular and demand no special treatment; if it grafts itself on a condition of cardio-vascular degeneration it enters into the category of cardiac pneumonias.

Pneumonia in the Intemperate.—Pneumonia, which is very frequent in hard drinkers, is often double, in any event it is likely to be very extensive. It has, says Fournier, a very rapid evolution, passing almost immediately to the second stage, and even to gray hepatization. It is frequently complicated with general prostration—adynamia, and ought to be considered among the most grave forms of pneumonia, even when there is no accompanying alcoholic delirium. Muscular trembling is one of the vouchers of the alcoholism, of which the arterial lesions (so general in

such cases) are the inevitable consequence; the latter contribute in great part to aggravate the prognosis.

The physician ought most carefully to explore the state of the heart and blood-vessels before instituting any formal treatment; he must neither enfeeble the heart by spoliative or depressant medication (bleeding and antimony) nor over excite it by alcohol. In alcoholic pneumonia, the frequency of which is constantly on the increase, I limit myself to prescribing tonic wines (such as Bordeaux), ale, coffee, concentrated broths and scraped beef. Cinchona in the form of the soft extract, associated with quinine or with digitalis cannot but be useful; the wine of cinchona always presenting a certain degree of inferiority relatively to the other preparations of cinchona.

Pneumonia in Diabetes.—Pneumonia, like tuberculosis—that is to say, the pneumonic bacterium, like the tubercle bacillus—easily takes possession of the diabetic patient, who seems to offer a most favorable culture soil to the microphytes; bacillary phthisis is one of the most frequent terminations of diabetes, and the same may be said of pneumonia. Every acute affection seems to act as do traumatisms; the pulmonary disease, whether it be broncho-pneumonia, or parasitic fibrinous pneumonia, often puts the life of the patient in danger; in fact the pneumonia in diabetes is always mortal, according to Durand Fardel. Bouchardat has seen instances of these rapid pneumonias where death has taken place in from twelve to twenty-four hours, or which have speedily terminated by suppuration or gangrene.

The practical question to know is whether such disasters are as much to be feared in the case of diabetics treated by an appropriate regimen, as in those who have been left to themselves and who have a fair semblance of embonpoint and health; it seems to me that the vulnerability of diabetic patients diminishes under the dietetic régime.

The Pneumonia of Polysarcic Individuals.—It is certain that corpulent persons, when attacked with pneumonia, are much less likely to get well than persons who possess the normal amount of adipose tissue. Polysarcic individuals often have fatty degeneration of the heart, and this state does not a little contribute to aggravation of the disease; favoring especially the asphyxiating dyspnœa.

Pneumonia in Cases of Inanition, Bodily Exhaustion, Cachexia and Anæmia.—The cardiac debility accompanying general denutrition and due to inanition, or to fatigue, or dilatation of the heart from general over-taxing of the muscular forces,—in short the decay of all the tissues and the lessening of the vital resistance in states of cachexia, impress on pneumonia an asthenic character which is natural and inevitable. All the tonics, all the most substantial aliments, are powerless before the general deterioration of the organism, and pneumonia comes to put an end to this physiological depravement and cachexia, even, as is often the case, without the temperature being appreciably elevated. (Lépine.)

The pneumonia of the chloro-anæmic is infinitely less formidable. Twenty per cent. of such patients get well, on condition that their strength be supported by nourishing drinks.

CHAPTER XXXIV.

TREATMENT OF ACUTE CATARRHAL BRONCHITIS.

Treatment of Tracheo-bronchitis or Rheum.—Tracheo-bronchitis or rheum requires no other treatment than rest in a warm room, whose temperature should not be above 65° F., for if it be raised higher than this in order to promote perspiration, it may cause or increase febrile movement. Warm drinks should be freely allowed, but hot drinks, given for a diaphoretic intent, may be injurious by causing undue relaxation of the tissues and inducing fresh cold; I allude particularly to the *sweating ptisans*, elderblow tea, thoroughwort tea, etc., which I believe are apt to do more harm than good, favoring rather than preventing propagation of the bronchitis to the capillary tubes.

Treatment of Acute Febrile Bronchitis.—From the moment that the catarrhal bronchitis passes beyond the trachea and large bronchi, a serious treatment should be instituted. The presence of dry or moist and diffused râles, with or without dyspnœa or fever, necessitates the interference of the physician, particularly in the case of children and old people. At the two extremes of life acute bronchitis speedily gains the bronchioles and leads to the development of capillary bronchitis or broncho-alveolitis; an affection, benign at first, soon becomes a grave disease. Here we realize the usefulness of emetics, which are especially indicated in cases of extreme repletion of the bronchi by mucus in children and old men, with whom expectoration is impossible or difficult. Ipecacuanha fulfils this indication better than tartar emetic, which has been previously studied. The following section will be devoted to the consideration of ipecac.

Expectorants.—Under this name must be designated all medicines which have the true or doubtful property of facilitating expectoration by changing the secretion so as to render it more liquid. We find in this category kermes mineral and the diaphoretic antimony, polygala senega, iodide of potassium,[1] and chloride of ammonium, which possesses certain advantages.

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[1] [In vol. i (Bacillary Phthisis) the author devotes a long chapter to the consideration of the physiological and therapeutical properties of the iodine preparations with special reference to their uses in pulmonary diseases. Their effect in stimulating the secretions of the respiratory passages in bronchitis, asthma, and especially the dry catarrh which marks the onset of tuberculosis is very remarkable. Under the influence of small doses of iodide of potassium, continued for a number of days, the secretions augment in the bronchi, and are of marked fluidity, so as to be easily expelled by coughing, and there is besides "a real excretion of morbid products which accumulate in the mucous cavities" (B. P. page 446). The anti-dyspnœic properties of iodine have been recognized for several years, being first shown by Prof. Sée in a memoir published in 1879; thirty or forty grains daily of iodide of potassium singularly facilitating respiration in the dyspnœa attending all forms of bronchitis and emphysema. While the iodides in concentrated solution are bactericide, there is no evidence that in medicinal

Narcotics.—Narcotics should be reserved to combat certain symptoms such as pain, dyspnœa, cough; apomorphia, which is a derivative of morphia, presents some similarity of action to the former, though having more affinity with the emetics.

Ipecacuanha.

Preparations. Doses.—Ipecacuanha is the root of cephælis ipecacuanha, which besides emetia, the emetic principle, contains tannic acid, and a glycoside (ipecacuanhic acid), starch and other substances of no account.

Ipecacuanha is prescribed in various ways, and in widely different doses, according as we wish to obtain vomiting, or only an expectorant effect; when we desire the latter we give small doses of the infusion. One gramme (15 grains) of the powder is steeped in eight fluid ounces of water and strained; of this a dessert-spoonful may be taken every two hours. The emetic dose is much larger; it varies from five grains to fifteen when the medicine is given in the form of powder; in children from two to six years of age the dose is from one to four grains, and to older children the dose would be twice or three times as much. The syrup is the form most often used and contains one part of ipecac. to one hundred parts of syrup; it is given in doses of one or two teaspoonfuls with or without the addition of a little powdered ipecac.[1]

Emetia, the active principle, is a colorless alkaloid soluble in concentrated alcohol, insoluble in water. Its physiological effects are sufficiently striking. In the frog one sixth of a grain suffices to paralyze the central nervous system and diminish the contractile properties of the

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doses they favorably influence in this way any pulmonary diseases of microphytic origin. Chloride of ammonium (sal ammoniac) is much used, especially in the country, as an expectorant in acute and chronic bronchitis; under its use the expectoration loses its irritating glairy character, and becomes more fluid. In old people, when there is lessening of the secretions, 10-grain doses should be given three times a day, dissolved in a teaspoonful of elixir of calisaya, and added to a small cupful of water. This salt should never be given unless largely diluted. Dr. H. C. Wood (Therapeutic Gazette, Jan. 1885) prefers muriate of ammonia to both squills and senega—"Like all ammoniacal preparations, this must be given at short intervals to maintain any constancy of effects. The action of the single dose can scarcely last more than two hours. Its acridity and disagreeableness may be somewhat covered by putting glycerine into the mixture. It must also be remembered that given in very large amounts all ammonia salts are capable of acting on the crasis of the blood as alkalies and of causing great vital depression."

A favorite preparation of Dr. Wood's, which he says has been tested for many years and found to be the most reliable and efficient sedative cough mixture which he has ever used, is the following:

℞ Potass. citrat. . . . . ℥ i.
Suc. limonis. . . . . f℥ ij.
Syr. ipecac. . . . . f℥ ss.
Syrup qs. ad. . . . . f℥ vi.
M.—S. A tablespoonful four to six times a day.—Trans.]

[1] The syrup of ipecac., according to the U. S. Ph., is made by taking two fluid ounces of fluid extract of ipecacuanha and adding it to thirty fluid ounces of syrup. The French Codex dissolves the alcoholic extract in water and then mixes it with syrup, but it is obvious that the U. S. plan is preferable, as it spares the continued heat requisite to reduce the tincture to dryness. One fluid ounce should contain the virtues of thirty grains of the powder. The emetic dose for an adult is about a tablespoonful, but more may be required. A teaspoonful may be given to a child a year or two old, and for an expectorant effect from two to twenty drops.—Tr.

muscles, without, however, destroying their excitability, unless at the moment of death.

A smaller dose—as one twelfth of a grain—suffices to interrupt the regularity of the heart's action till the ventricular contraction ceases altogether, and the heart is arrested in diastole.

In the higher animals the first effect is vomiting, which is brought about in a reflex manner as a result of peripheral excitation of the nerves of the stomach such as takes place when tartar emetic is given. Then there are observed constant violent movements of the intestine, with diarrhœa, simple or sanguinolent, accompanied by swelling and ecchymotic redness of the mucous membrane, as in poisoning by arsenic, antimony or iron. It is not by any direct action that these symptoms are produced; the poison is first absorbed.

The lungs are often found in a state of intense congestion, œdematous infiltration, and even red hepatization; this is especially noticeable in the hare poisoned by emetia. (Duckworth.)

The blood-vessels are paralyzed before the heart, as happens in poisoning by antimony or arsenic, which produce the same lesions and troubles of the intestine. The intra-vascular pressure is little by little reduced almost to nothing; it is then only that the heart is taken and the animal dies by cardiac paralysis.

Physiological Effects of Ipecacuanha. Emetic Action.—In the larger doses indicated above, ipecac. always produces vomiting owing to the emetia which is absorbed and reacts on the stomach on being eliminated; a part of the ipecac. is always expelled, which supposes also a direct action on the mucous membrane and its nerves.

Emeto-purgative Action.—A part of the ipecacuanha, especially when administered in the dose of fifteen or twenty grains, passes into the intestine, or by the intestine, according as it comes there directly from the stomach or from the blood after absorption. In such cases it produces the effects of an emeto-purgative.

Nauseant and Diaphoretic Action.—In fractional doses of a part of a grain, ipecac. produces a state of nausea, such as moreover always precedes vomiting. This pathological condition is characterized by malaise, a sentiment of faintness and muscular prostration, often by sweating with sensations of heat, by salivation, a secretion of buccal mucus, lastly, by a diminution in the beatings of the heart.

Have we here an instance of a special excitant action of the medicament at the same time on the vagus cardiac centre which slows the pulsations, on the œsophageal branches of the pneumogastric, and on the respiratory centre which determines the contraction of the diaphragm? There are strong reasons which favor this view.

These nauseating doses are what are especially utilized for an EXPECTORANT effect in bronchial affections, in order to facilitate the expulsion of accumulated secretions, and to change the character of the secretions, rendering the mucus more fluid. By this means also the irritation which provokes the cough is lessened, and the inflamed mucous membrane, which demands rest, is more easily restored to a healthy condition.

This expectorant action, like the state of nausea, is always attended with augmentation of the secretions, especially those of the bronchopulmonary mucous membrane, which lose their ropy tenacious character and become more watery.

Therapeutical Effects in Gastro-intestinal Affections.—Ipecacuanha has certain properties pertaining to the gastro-intestinal mucous membrane which have been utilized in therapeutics.

In the divers states called gastric and bilious, ipecac. is useful, but it is less employed than tartar emetic for emeto-cathartic purposes.

There is nevertheless a particular affection of the stomach where ipecacuanha in repeated doses produces good results. I refer to the mucous dyspepsias, and especially dilatations of the stomach, where the medicament is sometimes of striking benefit, as my clinical chief Mathieu has attested in a number of cases.

It is in nauseant doses, and in infusion, that ipecac. has been recommended and utilized with success in the treatment of DYSENTERY; this is the Brazilian method, much employed at Montpellier, and which resembles the contra-stimulant method by its depressant action on the pulse and heart.

Therapeutic Action in Bronchitis, Catarrhs, and Obstructions of the Respiratory Passages.—We find here occasion to put in use the three characteristic modes of action of ipecacuanha. It serves as an *emetic* with great advantages over apomorphia and tartar emetic, particularly in infancy, where there is always reason to fear the stibian diarrhœa and the nervous accidents which often follow apomorphia. As a nauseant, it surpasses tartar emetic, which too soon causes vomiting or not sufficiently, and in this event only operates as a contra-stimulant. In fine, as an expectorant it is vastly superior to kermes and the white oxide of antimony, stibian preparations relatively easy of administration with expectorant intent. Apomorphia is also inferior to ipecac. as an expectorant.

The Effects of Vomiting as Contrasted with the Effects of Coughing.—Why does vomiting expel more readily than coughing, the mucosities, membranes, etc., contained in the air-passages? In both cases there is an expulsive effort, and the thoracic cavity undergoes shrinkage. In coughing, however, the constriction of the chest is greater, because the diaphragm is forced upwards in a state of relaxation. In the act of vomiting, the diaphragm contracting lengthens the thorax, which undergoes shrinkage transversely, but the intra-thoracic pressure being lessened, the bronchi empty themselves more easily.

In coughing the glottis is tightly closed, and only dilates during the violence of the expiratory effort to allow the column of air to pass. In vomiting the glottis is open, the vocal cords are even relaxed; hence mucosities more easily clear the larynx by vomiting than by coughing.

EXPECTORANTS IN GENERAL.

Antimonials as Expectorants.—When we give the insoluble preparations of antimony indicated in a previous chapter (taking care by giving small doses wide apart to avoid the emetic effect) we ordinarily obtain in acute bronchitis, or in the recrudescences of chronic catarrhs, a more easy and more abundant expectoration. The physical signs change; the dry and sonorous bronchial râles transform themselves into humid râles, which become more and more liquid so as to facilitate the entrance of air into the lungs, and the respiratory murmur reappears in regions where it was temporarily suppressed by obstructions of the bronchioles. At the same time there speedily ensues a feeling of nausea which is accompanied by acceleration of the cardiac pulsations, and tendency to perspi-

ration; this consecutive diaphoresis physicians have sought to utilize for the benefit of catarrhal affections.

Expectorants with Emetia or Saponin for their Basis.—Polygala senega acts differently from the above, and owes its effects to saponin, an acrid principle similar to emetia, but less absorbable, and which acts only in large doses; saponin, like emetia, produces a state of nausea, a cough from the irritation which it occasions, and at the same time, an increase of the mucous secretion. We may, then, consider substances of this order as true, though feeble expectorants. Polygala, by reason of its slow absorption, presents certain advantages over ipecac., and may be continued a long time without producing the nausea which the latter causes, and seems to be better suited to the sub-acute or chronic catarrhs.

Medicaments which Increase Secretion; Muriate of Ammonia, etc.—There is a series of expectorants like sal-ammoniac which neither cause nausea nor diaphoresis; they simply augment the mucous secretions and make them more fluid. Chloride of ammonium, which stands at the head, acts differently from the other ammoniacal preparations, and presents great advantages in the treatment of chronic catarrh. It is the same with iodide of potassium, whose double fluidifying and expectorant action on the bronchial mucosities, as well as its respiratory stimulant power, we so much utilize in all pulmonary affections attended with dyspnœa.

Diaphoretics and Nauseants; Apomorphia and Jaborandi.

By the side of the expectorants, which are almost all diaphoretic, and before morphia, which is also a powerful sudorific, but an antagonist of bronchial secretion, we should place apomorphia, which is endowed with an emetic power like morphia, a nauseating property like ipecac., and is sudorific like jaborandi; it is employed to fulfil these three indications, but has not yet come extensively into use. Jaborandi acts in the same way; it is a diaphoretic and a very energetic nauseant, but is quite useless in the treatment of simple bronchitis.

Resumé of the Therapeutic Indications of Acute Bronchitis.

The therapeutic indications of simple bronchitis are restricted to feeble individuals, to young children, and to patients advanced in years; they are rather of a prophylactic kind, for what we want to do is to prevent the extension of the bronchitis to the capillary bronchi; we want also to prevent the accumulation of mucosities in the air passages. In children, ipecacuanha at first, then the white oxide of antimony, in doses amounting to fifteen grains a day, fulfil this double end.[1]

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[1] In this country the practitioner seldom sees any other preparation of antimony than tartar emetic, which is certainly unsuitable for expectorant purposes in the aged as well as in children. The white oxide, according to Dr. Bache, of the U. S. Dispensatory, deserves more attention than has been paid to it, being the least harmful of any of the antimonial compounds. It may be given in doses of a grain every two hours in syrup or mucilage.

Practically ipecac. answers all the purposes, in senile bronchitis, that can be fulfilled by the insoluble antimonials, and the same may be said of the bronchitis of childhood. The infusion of polygala with licorice and nitre, or carbonate of ammonia, constitutes an expectorant mixture which I have much employed and with advantage in bronchial catarrhs after the acute stage has passed.—Trans.

In aged patients it is still the insoluble antimonials which may be utilized, but on condition that the doses given shall be very moderate, and a prominent indication is at the same time to sustain the forces of the patient by nourishing drinks and alcoholic stimulants.

Treatment of Influenzal Bronchitis.[1]

Influenza is a specific affection which localizes itself principally in the air passages, and rarely fails, even in the benign forms, to disturb the nervous and digestive systems;—its grave forms manifest themselves especially by broncho-pneumonia, and demand the same treatment.

Specific Treatment. Quinine.—The causal (*i.e.* specific) indications are met by quinine, which is the certain remedy. Quinine not only antagonizes the epidemic cause, but it favorably influences all the manifestations of influenza, especially the nervo-motor and secretory troubles which accompany it constantly. The anti-parasitic action of quinine is well known, also the power which it has of arresting the diapedesis of leucocytes, and consequently the mucous secretion, which is the expression of this corpuscular transudation.

Therapeutics of the Gastro-intestinal Localizations. Emeto-cathartics.—Most physicians begin by prescribing an emeto-cathartic; this is naturally indicated when the disease outlines itself by that series of accidents which we have characterized under the name of gastric influenza, which really makes a part of the disturbances provoked by the fever. The antimonial or the ipecac. gives relief to the patient, and rids him of that state of nausea and malaise which marks the onset of the influenza; but we have not here an inevitable indication.

Treatment of the Sensory Motor Troubles. Quinine.—Whatever may be the degree and nature of the sensory motor disturbances, quinine is almost certain to stay their development: the neuralgic pains, the sensations of muscular fatigue, the physical prostration, all yield under the influence of fifteen grains of quinine, whether there be fever or not.

Treatment of the Bronchitis and the Broncho-pneumonia of Influenza.—As for the bronchial and broncho-pulmonary manifestations, they demand the same general treatment as the inflammatory diseases about to be considered.

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[1] The article Influenza was written with the co-operation of my clinical Chief A. Mathieu.

CHAPTER XXXV

TREATMENT OF CAPILLARY BRONCHITIS.

Broncho-pneumonia Compared with Ordinary Pneumonia as regards the Indications of Treatment.

Bronchiolitis, broncho-alveolitis, or broncho-pneumonia, as we have shown, recognizes two distinct origins, to wit, on the one part the propagation, altogether local, of the bronchitis to the alveoli, and on the other, the action of parasites which impress on the alveolitis its noxious character; this gives rise to two kinds of indications which are unfortunately more easy to state than to execute.

Comparison with Ordinary Pneumonia.—Broncho-pneumonia is readily distinguished from infectious pneumonia by its double etiology, as above indicated. Whether it be in the state of suffocative catarrh, of bronchiolitis, of lobular broncho-pneumonia, disseminated in the form of lobular nodules, or agglomerated so as to constitute what is called pseudo-lobar broncho-pneumonia, which definitively invades a whole lobe, like primary lobar-pneumonia, broncho-pneumonia takes on under all its forms, and always by reason of its complex causality, a strange paradoxical aspect; it follows an ill-defined course, and often after the most marked alternations,—now inspiring hope, now discouragement—comes to a fatal termination, despite the most enlightened medical treatment.

Comparative March of the Two Diseases.—What is the ordinary behavior of simple pneumonia? Its onset is marked by an intense hyperthermia, which at once prostrates the strongest man: then the fever heat remains about stationary, varying only several tenths of a degree during from six to nine days, pursues invariably a regular cycle as in the parasitic diseases, dominates during this time all the local manifestations, all the physical signs, then is suddenly extinguished, leaving the lung for one or two weeks to struggle with the inflammation which besets it; defervescence first appearing, then delitescence.

How is it now with broncho-pneumonia? It begins slowly, insidiously, under the form of a simple rheum, or a bronchitis *à frigore*, of an influenzal or rubeolic catarrh. There may be only a slight augmentation of fever to mark the passage of the simple bronchitis to pulmonary bronchitis; then the hyperthermia rises to 102° F. to 104° F., stops there or recedes, constantly oscillating under the supposed influence of congestions, of which it is certainly the cause rather than the effect. At other times the evil and the danger are revealed all at once by an asphyxiating dyspnœa, without the thermometer having given any warning; never is there anything like this in the fever of common pneumonia. Alveolar bronchitis is a disease with surprises, often of disastrous nature, and presenting none of the features of the undeviating cycle of parasitic pneumonia.

Individual Conditions, Ages, Contrasted.—A last feature of dissemblance is most striking. While infectious pneumonia smites indiscriminately adults as well as children, old age as well as adolescence; and while to the pneumonia microbe, as to the tubercle microbe all conditions of life are alike, it is not the same with respect to broncho-pneumonia, which selects particularly children of tender age during lactation and dentition; quite often those of from two to six years of age; more rarely older children; almost always spares adolescence and adult life; and falls with extreme intensity and frequency on the aged, in the case of whom it proves as fatal as in infancy.

Respective Influence of the Constitution on the two Diseases. Constitution and Force.—The fever and constitution play here a considerable part. Attention has been called to the facility with which in sickly or enfeebled children, in old men deprived of hygienic care, the most simple bronchitis propagates itself to the bronchioles and lungs. The adult resists the extension of the bronchial inflammation, and has, as has been said, "a good chest," *i. e.* of good conformation from the point of view of the bony framework, of the respiratory muscular apparatus, and of the bronchi, whose calibre is proportioned to the needs of the pulmonary ventilation. If you suppose the case of an individual who, although in full possession of the forces of maturity, has not sufficient muscular energy to aërate his lungs, or to clear the air tubes of the mucosities that encumber them, he belongs to the infantile or senile category, and is a good subject for broncho-pneumonia.

Local Predisposing Conditions.—The conformation of the thorax, and the previous state of the bronchi, exercise a still more decisive influence on the development of broncho-pneumonia.

Rachitism in children is one of the most favorable conditions, because it causes deformity of the chest, and thus constitutes a focus of stagnation for the air and the mucosities; scoliosis in the adult produces the same effect; the bronchitis thus easily transforms itself into broncho-pneumonia. Is anything like this seen in ordinary pneumonia? I do not know that simple fibrinous pneumonia has any predilection whatever for rickety children, and for hump-backed persons. It is not microbic pneumonia, nor even the tubercle bacillus, which attacks them most often, while they are constantly exposed after the least chill to bronchitis with all its consequences, including its steady invasion of the lungs.

Comparative Effects of the Micrococci on the Development of Pneumonia and of Broncho-pneumonia.—There remains a grave question to solve. The micrococci being the same in the two kinds of pneumonia, why do they develop here frank pneumonia and there bastard pneumonia, called pneumonia notha by the ancients? May we attribute these differences exclusively to the local state of the respiratory organs? Undoubtedly. The healthy and strong individual contracts the disease, that is to say breathes in the microbe, wherever it may happen to be met; as has been observed in places where people are congregated, in certain houses, in certain families where pneumonia prevails after the manner of a little epidemic; it is not necessary that there shall have been a previous bronchitis. On the other hand, broncho-pneumonia demands a previous condition, *sine qua non*, a prepared soil; there is no broncho-pneumonia without antecedent bronchitis.

Now the bronchi in catarrhal conditions offer a suitable culture medium in the mucus which obstructs them, and which the enfeebled infant

or the debilitated old man fail after a time to expectorate; the bronchitis gaining by proximity and continuity the capillary bronchi, the mucous exudation favors both the implantation and the multiplication of the microbe; we have here an additional factor facilitating the reproduction of the parasite, and the proof is that if bronchitis smites a constitution in a state of decline, at whatever age this may be, the primary phlegmasia transforms itself into a broncho-alveolitis, which seems at first simple, but which ends in becoming specific. Hence the cause of death in the broncho-pneumonic patient is double; he dies by obstruction of the bronchioles, and by the microbes; the pneumonic patient dies simply from the hyperthermia of microbic origin. At any age one may contract fibrinous pneumonia; persons do not take catarrhal pneumonia except after or in consequence of general or local debilitation.

The Different Species of Broncho-pneumonia Compared.

From this long comparison between the broncho-pneumonias and regular pneumonia this fact stands out clearly, to wit, that broncho-pneumonia is never primary; it results from a simple bronchitis by way of continuity; it oftener ensues as a sequel of the specific bronchites, such as attend influenza, where it is frequent; measles, which is so essentially broncho-catarrhal; whooping-cough, where it is more rare; diphtheria, which smites in a fatal manner all the respiratory passages, even to the bronchioles themselves. There is no need of insisting on the differing influence which these parasitic diseases exercise on the mucous membrane of the various branches of the bronchial tree—the gravity is at its maximum in diphtheria, it diminishes in measles and still more in whooping-cough, it is at its minimum in influenza. While the two extremes (diphtheria and influenza) are diseases which may recur with their pulmonary complications, measles and whooping-cough leave to the patient an almost certain immunity. The practical consequences from the point of view of the prognosis of the broncho-pneumonia, as well as that of the future of the patient if he gets well, are easily foreseen. One would *à priori* believe that simple broncho-pneumonia (*i.e.* derived from bronchitis *à frigore*) possesses always a certain benignity in comparison with the specific broncho-pneumonias; this is certainly the case, and the reason is apparent; all the broncho-pneumonias indiscriminately are parasitic; these specific phlegmasias are doubly so; this is why the latter are always more dangerous; the mortality of diphtheritic pneumonia is, so to speak, absolute; the physician is not quite so powerless in presence of the other forms, but that is all.

The different kinds of broncho-pneumonia, then, have a most marked influence on the prognosis and on the therapeutic results.

Treatment of the Broncho-pneumonias.

The comparative study of the curative indications in the bronchial and fibrinous pneumonias, has given us hints as to the grave difficulties against which the physician has to contend in the treatment. In passing in review the various medications which have a reputation or seem indicated in pneumonia, one very soon perceives that the greater part are inapplicable or even dangerous in the bronchial catarrhal forms; I enumerate the principal methods: 1. the antiphlogistics; 2. the contra-stimulants,

the emetics; 3. the expectorants and iodide of potassium; 4. the excitants, external and internal; 5. the antipyretics; 6. the dynamic medicaments; 7. expectancy.

Antiphlogistics.—Under this first head the opinion is pronounced: Cadet de Gassicourt, Picot and d'Espine discard altogether bleeding and leeches, even when the capillary bronchitis, accompanied by a violent congestion, invades both lungs and directly menaces the patient with asphyxia. Venesection and leeching not only do not diminish the asphyxia, but they augment it manifestly under the influence of the prostration into which the children rapidly fall.

Contra-stimulants. Emetics. Ipecacuanha.—The same proscription ought to extend to contra-stimulants. Even in the case of large children tartar emetic in large doses (three or four grains a day) may produce grave intestinal irritations, or even choleriform accidents, followed by mortal collapse. The antimonials under the rôle of emetics should be formally abjured; ipecacuanha advantageously replaces them. In the dose of from three to nine grains, the powder of ipecac. incorporated in half an ounce to an ounce of ipecacuanha syrup and given in divided portions every few minutes till free vomiting takes place, fulfils all the purposes of emesis. It is needless to repeat that emetics are of the greatest utility in promoting the elimination of the sputa which obstruct the air tubes, and that they should be given whenever this indication is urgent.

Expectorants. Kermes, White Oxide, Iodide Potassium.—When the bronchial element predominates, when the bronchiolitis has not already gained the alveoli, and there is no considerable collapse of lobules, expectorants, such as kermes, in doses amounting to two grains a day, or the washed diaphoretic antimony, from fifteen to thirty grains daily in divided doses, may render real service in augmenting and diluting the natural bronchial secretion, and in the case of larger children in favoring the expectoration of muco-pus. Iodide of potassium scarcely merits mention.[1]

Vesicatories and Revulsives.—When, on the other hand, the pulmonary element and the asphyxia tend to get the advantage, so as to interrupt the gaseous exchanges, neither expectorants nor emetics any longer find their applicability; then you must have resort to cutaneous or internal excitants. But the choice among the first is very difficult. The vesicatory, if it be of large dimensions, augments the fever, so Roger says, and in my opinion, it is likely to produce strangury. If, on the other hand, you have recourse to small flying blisters, you cannot help asking yourself if a real revulsive action is thereby obtained?—this is doubtful; in any event the small vesicatory is inoffensive and certainly preferable to mustard sinapisms or dry cups, which have no enduring effect, or to painting with tincture of iodine, which evidently acts only by absorption of the vaporized iodine.

Internal Excitants. Injections of Ether.—In grave cases it seems to me much preferable to resort to internal excitants, among which I may cite carbonate of ammonia or hypodermic injections of ether; it is then that alcoholic stimulants are urgently indicated.

Antipyretic Medication. Digitalis. Quinine. Baths?—It may happen that there is neither asphyxia nor exaggerated bronchial

[1] Hecht, Rieben, Gualdi (see Révue hebd. de thér. 1881, 1882, 1884, No 42) have counselled iodide of potassium in all the pneumonias. Three years ago I made a thorough trial of this medicament; it failed in all the periods of pneumonia, and of capillary bronchitis.

catarrh, but that the fever is the predominant and only serious symptom. This hyperthermia then demands treatment; to combat it the two best agents are digitalis (two or three grains of the leaves in infusion or maceration, to be taken during the twenty-four hours), and sulphate of quinine, of which five grains may be given in lavement. I have nothing but condemnation for the *bath*, and believe it more objectionable here even than in frank pneumonia. The warm bath is just the thing to augment the pulmonary hyperæmia, and the cold bath to increase the oxidations, *i.e.* the denutrition of the economy. There is no antithermic means more powerful and at the same time more slow than digitalis for children of from five to twelve years of age; it is especially efficacious in younger children, for whom the daily quantity should not exceed one or two grains.

Dynamic Medication.—This is the only medication which can properly constitute a system. Do not fear of aggravating the morbid process by daily administering to the child from one to two ounces of brandy well diluted in water. In prescribing it you are in no danger of augmenting the fever, for it is the opposite which takes place, nor can the local process be unfavorably influenced, though this will be the last to yield; the alcoholic potion will moreover prove a sedative rather than an excitant to the child The action of coffee is more doubtful.

Expectancy.—Combined with the dynamic action of alcohol, expectancy is the best of curative methods, on condition that it be accompanied by a nourishing regimen of milk and animal broths.

PART IV.

CHAPTER XXXVI.

PULMONARY GANGRENE.

Gangrene is a disease manifestly bacteric, which may invade the lungs in the general conditions of putrid fermentation.

Etiology.

It is important to know what are the micro-organisms of gangrenous processes, are they *special* or *common* bacteria?

The existence of BACTERIA OF SUPPURATION AND OF GANGRENE in surgical affections has been demonstrated with a wealth of details and descriptions of rare precision by Ogston of England (in 1881) and recently by Rosenbach (Micro-organisms, 1884). In the peripheral suppurations as well as in empyema (five observations), it was sometimes microbes in chains, which mycologists call streptococcus, or in clusters, which they designate under the name of staphylococcus. In the septic affections, the animated agents (according to Pasteur, Koch, Doleris), are not the sole producing causes of the septicæmia; you must also make large account of septic liquids [1] containing inanimate ferments, certain ptomaines (ptomainæmia), ammonia and sulphuretted hydrogen; [2] all these microbes, all these ferments, have the property of melting down tissues and causing them to suppurate; they are pyogenous.

In other cases there have been found certain fermentiferous micro-organisms, agents of putrid intoxication. König cites two cases; Ogston designates these microbes under the name of saprogenous; Rosenbach has succeeded in cultivating them, in inoculating them, and in producing a veritable gangrene.

Microbes of Pulmonary Gangrene.— Heretofore, in accounting for pulmonary gangrene mycologists have been contented with invoking the intervention of the leptothrix pulmonalis (Leyden) or certain monads which in fact are constantly found in the sputa in this disease and are the sure vouchers of all gangrene, *i.e.* of all putrid fermentation; the question is to ascertain if they are the only factors of putrid fermentation, or even if we ought not to attribute a predominant rôle to the pyogenous and septogenous microbes in general, and if, on the other hand, the alkaloids of organic disintegration, such as the ptomaines, have no part in this work of destruction. Whatever may be the solution of this complex problem it none the less remains a fact to-day gained to science, that the ferments of putrefaction and of gangrene have a *living* origin, as can be clinically verified.

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[1] Panum. [2] Rosenbach.

Definition of Pulmonary Gangrene.—We may clinically define pulmonary gangrene as the mortification and putrid destruction of the pulmonary tissue. To demonstrate that this is the true sense of this morbid process, it is needful to ascertain the mode of penetration of the septogenous microbes and the conditions of their development in the lungs; all the long and not easily understood series of causes called determining or predisposing also come into this confused etiological question.[1]

Immigration of Fermentiferous Foreign Bodies into the Lungs from Neighboring Organs.

Involuntary Introduction into the Bronchi of Alimentary Particles.—Alimentary particles swallowed the wrong way, or without consciousness, may penetrate the larynx and reach the pulmonary bronchioles, bringing with them the ferments of destruction; this is one of the most frequent causes of pulmonary gangrene.

This involuntary immigration of food fragments undergoing putrefaction or digestion has been noticed from all time in very young or debilitated children, in the aged and decrepid, who have arrived at a general paretic condition, and in enfeebled individuals generally, and particularly those who have some serious affection of the central nervous system.

Ghislain was a long time ago struck with the frequency of death from pulmonary gangrene in lunatics; all the alienists have recognized the truth of this observation, which is easily explained by the forced feeding and the use of the sound to which madmen are often obliged to submit, and by the difficulties of deglutition which they experience. Persons suffering from bulbar paralysis die of inanition by reason of loss of function of the glosso-pharyngeal and hypo-glossal nerves, or it may be, die of pulmonary gangrene; they can no longer swallow anything, or if they attempt to swallow, the alimentary matters are likely to stray into the larynx and penetrate the alveolar bronchioles, where they determine sphacelus, all the conditions of heat and humidity favorable for putrefaction existing in the pulmonary tissue.

In a more general and vague manner debilitations resulting from what has been called PHYSIOLOGICAL DEPRAVEMENT have been cited as causes of gangrene. In 1844 Mosing described an epidemic of pulmonary gangrene which attacked all the prisoners of a wretched penitentiary. This epidemic of gangrene has been called in question, and Mosing has furnished no proof in support of his diagnosis, and for

[1] "Fortunately gangrene of the lungs is not a common disease. But two cases have occurred at the Massachusetts Reformatory Prison for women since its establishment in 1877. At the Boston House of Industry, on Deer Island, no cases have been reported during that time, and but two have occurred at the Boston House of Correction—institutions which together represent about two thousand persons. * * * The disease is said to appear most often in the insane, in drunkards, in epileptics, and in children. A low state of nutrition, with weak heart action, is a predisposing cause. An interruption of the blood current, the passage of foreign bodies into the air passages, and traumatic influences may be numbered among the chief exciting causes. * * * The physical signs of infarction are so obscure that an early diagnosis is rarely made. Dullness on percussion over a limited area with an irritative cough, accompanied by a weak pulse and general malaise with pinched features, is usually all that is observed until the inflammatory fever arises and the breath becomes putrid."—Dr. Eliza M. Mosher, in New York Medical Journal, Aug. 29, 1885.

aught that appears in his report his epidemic may have been one of infectious pneumonia, such as often prevails in unhealthy crowded habitations, barracks, etc., and is characterized by phenomena called putrid. What is certain is that the lung frequently undergoes mortification as a result of contact with particles of food in the process of digestion.

Translation of Gangrenous Particles by way of Continuity.—Gangrenous fragments proceeding from the mouth or pharynx and containing the putrid bacterium, may directly penetrate the lungs. In children affected with noma death often takes place by gangrene of the lungs; it is the same when the sphacelus occupies the pharynx, or even when after operations practised in the bucco-pharyngeal cavity suppuration is established. Now what takes place in these cases? To solve the problem of this multiplicity of gangrenes a *gangrenous diathesis* has been imagined; this is a phraseology familiar to medical philosophy, but the reality is more simple. We have to do with the action of bacteria, of the leptothrix of the mouth or throat, which are set free from the sphacelated parts and penetrate the lungs, where they multiply and produce their work of destruction.

It is also easily understood how it is that in measles and whooping-cough, which furnish the largest proportion of cases of noma and pulmonary gangrene in children—we see again the influence, not indeed of a general debilitation, but of a virulent principle of the nature of a fermentiferous bacterium, which may so act on the mucous membrane and the buccal secretion, that the transportation of the putrefied products to the lungs may there produce a disorganization of the same kind.

Instead of gangrenous débris, often it is the products of the suppuration of neighboring organs, which are discharged directly or indirectly into the air passages and there produce gangrene. Volkmann has seen this as a sequel of caries of the petrous bone.

Penetration of Virulent Products by way of Contiguity.—The virulent products may reach the respiratory organs by way of contiguity. Thus the pus may come from an abcess of the œsophagus, from encephaloid cancer of the œsophagus, from cancer of the cardia. Thus it is also that suppuration of the pleura, abscesses of the lungs, of the vertebræ, of the sternum may empty themselves into the bronchial cavity and cause mortification of the pulmonary tissue.

Immigration of Gangrenous Emboli.

Bacteriferous fragments in general do not always reach the lung from other organs by way of contiguity or continuity; the blood itself may be the bearer of emboli, and cause them to lodge in the lung.

Fibrinous Embolism and Thrombosis.—Now among these emboli, some formed by coagulated blood may obliterate the pulmonary capillaries, cause the veins of the obstructed lobule to burst, and provoke an interstitial hæmorrhage—a pulmonary infarctus,—but they do not give rise to gangrene; the hæmorrhagic infarctus itself may compress the capillaries of the neighborhood, and ischæmiate a considerable territory without nevertheless going so far as to produce gangrene.

Gangrenous Emboli.—But the blood often serves as vehicle to emboli of septic or gangrenous nature, which are carried to a department of the circulation naturally limited, and there form a circumscribed gangrene. Now these septic emboli present themselves under the following

circumstances: When as a result of the eschars following prolonged decubitus, or osseous suppurations, or puerperal lesions, there develops a local gangrene, then a process of necrosis at a distance, it is because from the point primitively gangrened there is detached a putrefied fragment which has penetrated a vein and thence has found access to the lung. In the case of multiple gangrenes, where in explanation a gangrenous diathesis or even a metastasis has been so often invoked, that is to say a displacement of the gangrene from one point to another, the only real displacement is the transportation of gangrenous and bacteriferous débris, which find in the lungs lodgment and all the conditions favorable to putrid fermentation.

Local Conditions of Sphacelus and of Fermentation.

We have just enumerated the two certain admitted modes of the production of gangrenes; other mechanisms have been alleged, such as gangrene by obliteration of the pulmonary vessels, gangrene by excess of inflammation, etc., but most of these causes are disputable, and are generally relegated to the rank of predisposing circumstances.

Vascular Obliterations by Thrombosis.—Theoretically gangrene ought to ensue whenever there is obstruction of the vessels and thrombosis; we know, however, that the pulmonary vessels are of two orders—the bronchial arteries and their dependencies, which minister to nutrition, and the pulmonary arteries, whose office is functional. Now the latter are all terminal arteries, that is to say, disposed in such a manner that neither congestions nor (for a stronger reason) gangrenes can localize themselves, and a gangrene from an obstruction of these vessels would have to be general. As for the bronchial arteries, neither their obstruction by clot, nor their compression by hæmorrhagic infarctions are sufficient to arrest the life of a limited part of the lung; we know no instance of obstruction or gangrene of this kind.

Pneumonias.—Since the time that Laennec described pulmonary gangrene, all writers have spoken of pneumonias ending in gangrene; now this termination is possible in the broncho-pneumonias (Cornil and Ranvier); it is not so in the true pneumonias, as Lépine has well remarked. It has not, in fact, been demonstrated, when hepatization has been found around a gangrenous focus, that the inflammation was primordial; it is the contrary which is generally true, and septic products easily determine inflammatory reactions in the lung. Despite the uncertainty of the facts, theories have not been wanting to explain them. Sometimes the mortification was attributed to an excess of inflammation, sometimes to a sort of strangulation, comparable to what is observed in phlegmons; now the most intense, the most extensive, the most profound inflammation in an individual otherwise healthy, cannot produce gangrene; the comparison with phlegmon is especially unfortunate, for phlegmon is itself a parasitic production, and the gangrene which follows it is itself a putrid fermentation.

The post-pneumonic gangrenes develop exactly like those which succeed fetid or septic bronchitis, dilatations of the bronchi, or even tuberculous cavities (where gangrene is, however, very rare); in all these cases the respired bacteria find a favorable culture soil; this is what is observed particularly in certain bronchites.

Fetid and Gangrenous Bronchites.

Gangrenous Bronchitis.—Fetid bronchitis has been considered sometimes as the result of a special gangrenous lesion limited to the extremities of the bronchi (Briquet), and sometimes as the effect of a putrid decomposition of the products of mucous secretions; it may take place in both ways, as we shall now see. The gangrene rarely rests limited to the bronchioles, it passes to the pulmonary tissues; conversely, when the gangrene invades the lung it does not always spare the capillary bronchi.

Fetid Bronchitis.—We are not concerned here with gangrene of the tissues but with the products of putrefaction, which develop in the bronchi when chronically inflamed or dilated; when the mucus sojourns a long time in these cavities, it constitutes a culture liquid most favorable for the reproduction and development of the bacteria which penetrate the lungs.

In a case of putrid bronchitis which we published three years ago (Gaz. Méd. 1881), and which terminated in death, the autopsy did not reveal the least necrotic alteration of the bronchi or lungs; it was simply an instance of fetid bronchitis; death, which took place in the midst of a general prostration with complete decoloration of the teguments, and excessive emaciation, was only explicable by the resorption of the putrid products and by the production and absorption of the ptomaines which so often develop in dead compounds.

Putrid Bronchiectasies.—Here we have only to do with the decomposition of the sputum; these are not cases of gangrene. (See the observations of Dittrich and Lasègue.)

Local Irritants, Septic Gases.

Among the septic principles which act directly on the lungs, we must mention sulphuretted hydrogen gas and ammonia, which are evolved from sewers and receptacles of night soil; pulmonary gangrene has been quite frequently observed in scavengers. Jaccoud has also cited a case of gangrene following the breathing of the gases resulting from the combustion of picrate of potash.[1]

Traumatisms.—I finish this series of local irritations by the traumatisms. Leyden, Bucquoi and Hanot have reported cases of pulmonary gangrene by contusion and compression of the thorax.

Foreign bodies which accidentally penetrate the bronchi without themselves having any virulent character may determine gangrene; cherry-stones (Jaffé), a fragment of bone (Leyden and Strumpel), grains of wheat, have been introduced into the bronchi by a false movement of deglutition, have even gained the bronchioles and produced a traumatic necrosis, evidently due to the direct destruction of the pulmonary tissue.

General Septigenous Conditions. Toxæmic, Glycæmic, Alcoholic Gangrenes.

An occasional cause of gangrene has been said to be alterations of the blood, but thus far none are known which are able to produce either putrefaction of the pulmonary tissues or any kind of gangrene.

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[1] "In 1869, after the accident of the Place de la Sorbonne, I had the misfortune to see one of my externes succumb to multiple pulmonary gangrene resulting from the inhalation of gases arising from combustion of picrate of potassa." Jaccoud, Pathologie Interne, t. ii., p. 38. 1875,

Diabetic Gangrene.—The frequency of gangrene in the diabetic seems to give support to the supposition that glycæmia constitutes precisely one of those predisposing alterations of the blood; it cannot in fact be denied that this condition offers a culture soil singularly adapted to the pullulation of bacteria, but we know nothing more.

Alcoholic Gangrene.—Alcoholic poisoning, which is equally a condition favorable to the production of gangrene, can no longer be considered as thus favoring it by blood changes; it is the vascular sclerosis which seems here to play the most important rôle in producing impediments to the circulation, stasis of the blood in the vessels of the lungs, and the consequent development of bacteria.

Resume and Explanations.—To sum up; the most evident causes of pulmonary gangrene are the direct penetration of bacteria and more rarely embolism. Theoretically, one would suppose that the bacteria penetrating more directly by the right bronchus, which is the largest, would be more likely to find a lodgment in the medium-sized bronchi; and this is exactly in accordance with data which statistics have furnished. It is in fact very easy to interpret the marked frequency of gangrenes on the right side, their predominance in the middle lobes, their rarity as diffuse and bilateral gangrenes, by the physiological considerations just mentioned; all the statistics of Lebert are in accord with the previsions of Laennec on this point. It is the same with respect to the very limited mortifications of the inferior lobes, or the very superficial sub-pleural gangrenes, whose rarity is proportioned to the rarity of their causes; I have reference particularly to gangrenes from septic embolism, which limit and localize themselves in such an apparently exceptional manner, while being etiologically only a sequel of the natural effects of emboli in the superficial and inferior pulmonary vessels, the ordinary seat of these migratory clots.

Effects of the Gangrenous Process on the Lung.

We may follow not only the localization and march of the gangrenous process, but also analyze with exactness the effects which it produces on the texture of the lung.

Anatomical Forms.—Laennec has already testified that the invasion of the lung may be considerable, the gangrene being diffuse; that moreover in this case, the part altered is transformed into a greenish black or greyish mass impregnated with an infectious sanies, that this mass presents a friable consistence, seems as if macerated, and even passes gradually, without precise delimitation, upon the sound parts and shows a great tendency to spread. This is, as we already know, and for the reasons already indicated, the rare form of gangrene.

The circumscribed form, which is the most common, presents itself often in multiple foci; this is the explanation: when the upper lobes are alone affected at the onset, the sanies which flows from them falls into the bronchi of the inferior lobe and forms there secondary foci. In all these centres the mortification follows, as Laennec shows, three phases.

In a first phase there forms a sort of greenish black eschar, which is not slow to undergo disintegration and softening at its circumference, and to separate itself from the surrounding parts as a sort of sequestrum, which henceforth occupies a cavernous focus.

The formation of the cavern constitutes a second phase of the mortification; the gangrenous excavation, which is of an irregularly roundish form,

of very variable dimensions, presents divers interesting peculiarities from the point of view of diagnosis and prognosis. It offers an internal surface which is uneven, velvety, as if torn, and full of a granular flocculent dirty grayish fluid, of a putrid odor, just such as a patient is destined to expectorate. In this cavity open certain bronchi with clean cut extremities; these tubes may possibly be competent, when communication is very free, for removal of the entire eschar, unless the latter softens and passes to a fluid state before expectoration is established. A third peculiarity is this: All the vessels comprised in the gangrenous region become obliterated by thrombosis and by inflammatory alteration of the endothelium; they may nevertheless be invaded by the gangrenous ulceration, become lacerated and furnish an extravasation of blood, with hæmoptysis. A last circumstance to note is the circumscription of the focus by hepatized tissue or interstitial pneumonia, and the inward delimitation by a pyogenic membrane causing the cavity to collapse, and its walls to come together; perhaps even to heal. When the connective tissue capsule is wanting, the foci spread, and if they are superficial they gain the pleura, which becomes the seat of a purulent pleurisy, or undergoes perforation, so as to form a pyopneumothorax. Authorities have also mentioned perforations of divers neighboring organs, and a discharge of sanious pus through the diaphragm, œsophagus, or mediastinum.

CHAPTER XXXVII.

CLINICAL STUDY OF PULMONARY GANGRENE.

From the point of view of diagnosis, we must make a fundamental distinction between the gangrenes with putrid expectoration, which alone can be recognized, and those which only reveal their presence by the physical signs of a focus of induration, or of a cavity; this is why it is important to know well the nature and composition of gangrenous sputa.

It is the expectoration which constitutes the most important diagnostic character; if this fail us, that is to say, if the sputa are arrested at their period of elimination by the excessive swelling of the bronchial mucous membrane, or by compression of the bronchus, or if the patient has not the strength to expectorate, one may be in danger, at least for a time, of overlooking the gangrene, and of confounding it with other pulmonary lesions which present the same physical signs.

Physical Characters of the Sputa.—The odor of the sputa is so penetrating, and recalls so completely that of putrefaction, that it is difficult to mistake it; the air breathed out by the patient is infected to such a degree that the fetidity makes itself perceived at a distance, impregnates the atmosphere, and renders the habitation intolerable.

Ordinarily more or less diffluent, the sputa are very abundant, and may even exceed 1000 cc.; the expectoration generally comes in floods, and several times a day the patient renders enormous mouthfuls of the infectious liquid.

Usually of a grayish green color, or of a brick-dust color if they contain blood, the sputa separate in the spit-cup into three strata; the upper layer, which is frothy, greenish yellow, opaque, contains isolated flakes of pus and mucus. The middle part is more fluid and is like serum. The lower stratum, which is sanious, and formed almost exclusively of yellowish pus, contains numerous lumps of variable size, plugs, and shreds of lung tissue.

Micro-biologic Characters of the Mucous Plugs. Mycoderms. Infusoria.—The bronchial plugs, which are also met with in fetid bronchitis, and which by reason of their weight always sink to the bottom of the spit-cup, are constituted almost entirely of masses of very fine filaments and spores of the *leptothrix pulmonalis;* these lumps in the fresh state contain also two sorts of infusoria—the *monas lens* and the *cercomonas;* it is these fungi and infusoria which are the true factors of putrid decomposition.

In the midst of these masses of fungi, there are also found cells which in old sputa contain numerous fat droplets; at a stage more advanced there appear, besides liquid fat, crystals and needles of margarine. All these masses of parasites, these fragments of amorphous or crystallized fat which constitute the *corps du délit* (offending body) come evidently from the bronchi, for after death we notice the same identical products in the

bronchial tubes of medium and small calibre, which belong to the gangrened pulmonary lobules; at the same time it is difficult to say what are the bacteria of putrefaction. Leyden and Jaffé think that the leptothrixis principally concerned in this destructive process.

Fragments of the Lung. Elastic Fibres.—Another characteristic, no less important, of these sputa, is the presence of débris of the pulmonary tissue, easy to recognize by their grayish or blackish color.

In this détritus elastic fibres are quite frequently distinguished; Traube has affirmed that they almost always dissolve in the peptones, and are rarely met with in the sputa.

Chemical Composition.—Chemically examined, the sputa are found characterized by the presence of all the products which are formed by the putrefaction of organic substances; to wit, tyrosine, leucine, sulphuretted hydrogen, butyric, valerianic acids, etc. It will be incumbent henceforth on the clinician to search also for the alkaloids of decomposition, the ptomaines.

In a certain number of cases the expectoration is mixed with BLOOD; the progress of the gangrene may cause ulceration of the vessels and hæmoptysis, or at least, extravasation of a small quantity of blood.

The sputa of putrid bronchitis present the same microbic characters, the same chemical composition, as the expectoration of pulmonary gangrene; there is but one difference, which is readily foreseen: the sputa in the former case contain no débris of pulmonary tissue, and still less, no elastic fibres; Rosenstein also thinks that here the leptothrix does not constitute the agent of putridity, but rather the thrush fungus, the *oïdium albicans!*

Diagnostic Value of the Expectoration.—The expectoration may be present from the first day, as well as the *fœtor oris*, which sometimes even precedes it; in general it shows itself as soon as the gangrenous mass softens, and communicates with a bronchus; when the bronchial tube is obstructed, it disappears, to return as soon as the bronchial cavity becomes permeable. There is never any expectoration when the gangrened lobe or lobule is cut off from all communication with outside, as takes place when the gangrene is at the periphery (cortical), as Corbin and Bucquoi have shown.

SEMEIOLOGICAL CHARACTERS OF THE PULMONARY GANGRENES IN GENERAL.

Value of the Physical Signs.—The gangrene begins sometimes suddenly, like an acute pleuro-pneumonia, the appearances of which it takes on, before revealing itself by the expectoration; sometimes it declares itself slowly by a cavernous focus, in taking from the latter its semeiology; sometimes, finally, as happens when the seat of the gangrene is cortical, or pneumo-pleural, it pursues its entire course without revealing its hidden nature by the least characteristic sign.

Diagnostic Value of the Adynamia and Digestive Troubles.—But if the physical examination does not always give definite hints in the way of diagnosis, most important indications are furnished by the adynamia which characterizes all the gangrenes indiscriminately. Let no one here be deceived into thinking that this prostration is the effect of previous debilitation, such as is often to be reckoned among the antecedent and

predisposing conditions of the gangrene. The adynamia is of the nature of profound muscular and nervous enfeeblement and lassitude; a general exhaustion of the vital forces manifesting itself with the most marked cachetic appearances in individuals heretofore healthy.

The digestive organs suffer the repercussion (*contre coup*) of this exhaustion, and by their disturbances tend to augment it; the ingurgitation of sputa determines profound anorexia, vomiting, dyspepsia and diarrhœa. All these grave phenomena, which fatally compromise life, ought to be taken into consideration from the point of view of diagnosis.

Diagnostic Value of the Functional Troubles.—In a remarkable description of the gangrenes by Bucquoi, and reproduced by Strauss, one cannot fail to be struck with the contrast between the functional troubles and the physical signs in gangrene of the lungs. Gangrene produces, especially when it is cortical, pain of the most intense kind, an incessant painful cough, and a dyspnœa which is proportioned to the extent of the lesion. These symptoms constitute precious elements of diagnosis in suspected sphacelus of the lungs.

The Principal Types of Pulmonary Gangrene.

The onset of pulmonary gangrene is marked by symptoms which may be referred to three principal types, which in fact characterize the whole course of the disease; the first takes the form of acute pseudo-pneumonia; the second of excavation, with slow march; the third of gangrenous pneumo-pleurisy.

First Type.—Pneumonic Gangrene.—In a certain number of cases the disease announces itself by a violent pain in the side, dyspnœa, viscous sputa, sometimes sanguinolent from hæmoptysis (Behier, and Hardy) and a severe cough. The physical signs are as follows: subcrepitant râles limited to one region, which presents a slight degree of dullness and a diminution more or less marked of the respiratory murmur, later of the tubular souffle. The characteristic feature is the profound alteration of nutrition and of the muscles; although the fever may be neither very intense nor continuous, you are struck with the pallor and lividity of the face, the alteration of the features, the prostration which scarcely permits any movements, the state of dreamy agitation without delirium, the lipothymic tendencies, the feebleness of the pulse, and the dryness of the integuments, alternating with cold and clammy sweats. These patients die rapidly and surely.

Second Type.—Cavernous Form.—At other times the disease comes on slowly without fever and manifests itself by general denutrition and depression, by the earthy color of the countenance, and it may be by a short intermittent cough.

Then you note the signs of a cavity, the tubular or amphoric souffle, accompanied by gurgling sounds and dullness on percussion; you might be led to suppose there was a bronchial or tuberculous cavity, but soon the fetid odor of the breath, then the putridity of the sputa, reveal the origin of the evil, which is no less deadly, although more slow, than the pneumonic type.

Third Type.—Pneumo-pleural Gangrene, without Expectoration.—The third type remains almost always latent. It is, as Fournier says, a gangrene, which instead of marching towards the bronchi takes its

direction towards the pleura, which undergoes perforation by the sanious pus, and becomes the seat of a purulent or gangrenous pleurisy, or of a pneumothorax. It is necessary to distinguish these cases from the old pleurisies whose effusion becomes putrid and which are capable of getting well by empyema; it is necessary also to distinguish them from cases of pleurisy followed by mortification of the lung. (Observation of Dolbeau, reported by Millard.)

Diagnosis by Causes and by Signs.

If one depends solely on the signs, that is to say, on the types which we have described, the diagnosis will sometimes present insurmountable difficulties. All, in fact, will hinge on the putrid expectoration. As soon as you have detected this, then all that you have to do is to distinguish it from expectoration that is simply fetid. Here etiology comes to the aid of the clinician, and enables him to make the distinction. The gangrenous nature of the expectorated matters being recognized, there will be a second question to clear up, to wit, if the gangrene has any discoverable cause, and what is this cause; this constitutes the etiological diagnosis of the true gangrenes. The third problem is of a more complex and difficult nature; the demonstrative evidence (expectoration) is wanting, and we are quite in the dark as to the origin of the general symptoms and physical signs; the gangrene is latent, and must be discriminated from the other pleuro-pulmonary lesions. Now it may happen that the etiological considerations will throw some light on the subject; this will be the etiological diagnosis of the latent gangrenes.

I. **Diagnosis of the False Gangrenes.**—The fetid expectoration presents a great similarity to gangrenous matters; it differs only by the absence of fragments of the pulmonary tissue, and of elastic fibres from the lungs, of which the one is constant and the other frequent in gangrenous patients. False gangrene is observed under the following conditions:

(*a.*) **Fetid Bronchitis with Chronic Broncho-catarrh.**—These cases are generally grafted upon chronic catarrh, and constitute only an incident in the course of the affection, which is sometimes grave by reason of *ptomaine* poisoning, but may disappear spontaneously or under treatment; it is without doubt to these pseudo-gangrenes that we are to refer the most of the pretended cures of true gangrene; curative effects being attributed to the antiseptic medications, where the diagnosis was erroneous. To avoid this mistake it is important also to be able to determine the nature of the bacteria found in the fetid sputa; are they of the same kind as the bacteria of gangrene? This is a question which I put, without being able myself to answer it. It is the more important to obtain all the light possible from the microscopical examination of the expectorated products, since the physical examination is generally incapable of enabling us to discern fetid bronchitis from gangrene; both the one and the other reveal themselves often only by large coarse râles without any sign of condensation of the pulmonary tissue, that is, without any dullness or souffle. There will be nothing left, then, after a thorough microscopic study of the expectorated matters, but to take into consideration the absence of the signs of adynamia; but all this constitutes only an uncertain basis of diagnosis.

(*b.*) **Bronchiectasis with Fetid Products.**—In the present case

you notice the signs of a cavity, which are more or less obscured by the signs of pulmonary induration, that is to say, of the sclerosis which invariably attends bronchial dilatations. It is the same with the gangrenes, which manifest themselves by the signs of an excavation covered over by inflamed parenchyma—a hepatized or sclerosed envelope. The distinction between these two affections rests only on the histology of the sputa and the chronicity of the bronchiectasis.

(*c.*) **Tuberculous Cavities.**—Here the presence of the bacilli will remove all doubt.

(*d.*) **Pulmonary Abscesses.**—Pulmonary abscesses may open into the bronchi and discharge pus mingled with blood, but rarely fetid; if they present a putrescent odor, one may still distinguish these purulent and fetid sputa by the constant presence of elastic fibres, which are often wanting in gangrene.

(*e.*) **Pleural Vomica.**—A suppurative pleurisy, free or encysted, general or interlobar, often opens into the bronchi, which then furnish an abundant expectoration of several ounces of pus become fetid from decomposition during its sojourn in the pleura. This pleural vomica may continue to discharge pus and simulate a gangrenous cavity.

But here the physical signs scarcely admit of doubt; the absolute dullness, the tubular blowing, the œgophony, the absence of vibrations, are never found together in gangrene, and are always more or less associated in pleurisies, however partial.

II. Etiologic Diagnosis of True Gangrene.—True gangrene being distinguished from the false, it only remains to verify and complete the diagnosis by the etiological circumstances which gave rise to the gangrene. In this connection you should recall to mind the frequency of gangrene by immigration of alimentary particles, putrid fragments, etc., in feeble individuals, in children, old men, the insane or demented, and in drunkards, in fine the possibility of the gangrene being diabetic should be entertained.

Diagnosis of the Latent Gangrenes.—Latent gangrene, which is generally cortical, is inevitably confounded with pleuro-pneumonia. The intensity of the pain, of the cough, of the dyspnœa, does not suffice to dissipate the doubts, so that it will not do to forget the determining conditions of latent gangrene, among which I must mention especially gangrenous emboli from a sphacelated part of the periphery, or from some one of the viscera; it is these specific emboli which ordinarily form the circumscribed gangrene of the pulmonary sub-pleural cortex; despite all, the diagnosis is hardly ever verified till after death.

Prognosis and Treatment.

There are gangrenes which lead rapidly to death. These are the gangrenes which are diffuse in respect to their destructive lesions, as well as those which are acute in respect to their evolution; there are gangrenes which are neither diffuse nor acute, but which are nevertheless fatal: I refer to those which have an embolic origin, or to those which depend on invasion of the lung by foreign bodies or putrid substances, and this class comprehends a great number of cases. What, then, are those which may get well? It has been affirmed that it is of little consequence what the cause may have been; that the curability is limited to individuals who are

not too debilitated, to cases which have a slow course, so slow in fact that it may be possible for the powers of the organism to establish around the gangrenous cavity a demarcating inflammation, and in the cavity, a pyogenic membrane, susceptible itself of retraction and cicatrization and of closing the putrid cavity. In fact there have been found at autopsies vestiges of these phlegmasias and of these membranes; but they were *autopsies*, bear in mind, and autopsies which prove clearly that one may die in the midst of the curative endeavors of nature. It is not at all doubtful that cases of recovery are to be referred, generally, if not always, to the pseudo-gangrenous forms.

Therapeutic Indications.—There exists but one etiological indication, and that consists in the prevention by antiseptic means of the development and multiplication of the bacteria of putrid fermentation, and especially in the disinfection of the sputa, which not being expectorated, may be sucked down by the bronchi which still remain healthy. One other indication, which may be called an individual indication, is to sustain the forces of the patient, always so gravely affected previously or in consequence of the disease, in order to enable him to struggle against the progressive invasion of the sphacelus.

Antiseptic Medication by Inhalation.—To reach more surely the micro-organisms in the bronchi, recourse has been had to inhalations. Stokes in 1878 published a case of recovery from gangrene by chlorinated fumigations, which have never been essayed since that time.

Fumigations with Turpentine.—In 1852 Skoda recommended fumigation with spirits of turpentine poured upon boiling water, and aspirated from an inhaling apparatus. These fumigations have often been employed, though with very doubtful success.

Phenic Acid.—During the last few years, clinicians have made use largely of inhalations of phenic acid in solutions of from 2 to 5 per cent., these inhalations being repeated from two to eight times a day in séances of from five to ten minutes. Curschmann has thought to render the aspirations more efficacious by means of a respirator of his own invention which sets free the phenic vapor in such a way that the patient is obliged constantly to breathe it.

Whatever may be the method employed, if it is even certain that the phenol is absorbed, it is very doubtful if it reaches the diseased part, the antiseptic action not being direct; however, it none the less presents grave inconveniences and often real danger. In fact the toxic effects are not slow to manifest themselves, and one of the first symptoms is the olive green color of the urine; when it comes to this, it is necessary to stop and substitute some other medicament, such as the terebinthinated vapors.

Boric Acid, etc.—Attempts have been made, in order to obviate this danger, to employ in a similar manner a solution of boric acid or of borosalicylic acid, or of bromine water, but thus far no satisfactory results have been attained.

Internal Antiseptic Medications.—Phenic Acid.—Again recourse has been had to phenic acid, which has been used internally in doses of from 4 to 15 grains by Leyden; it is as difficult to believe in the efficacy and innocuousness of this method as in the favorable effects of the phenic inhalations.

Sulphate of Quinine.—In my own experience, it is the sulphate of quinine which gives the least bad results, on condition that it be pre-

scribed in doses of twenty grains a day and associated with cinchona or gentian.

Tonic and Supporting Treatment. Animal Food and Milk.—It is of chief importance to carefully direct the dietary regimen of the patient, in order to sustain the flagging vital forces. For this end milk in conjunction with raw meat should be ordered, and without stint; this is the kind of food which is the best supported by the stomach, always deranged in its digestive powers. The medicaments called tonic, such as cinchona wine, have no effect, because they contain no active principle unless it be the tannin in them, which the patients cannot tolerate; it will be better to give coffee or red wine, which contains but a small percentage of alcohol. The excessive or prolonged use of alcohol has been rightly considered as a means of favoring putrid fermentation; alcohol itself ferments and does not maintain the forces beyond a certain point and that temporarily.

Resume.—Sulphate of quinine, a diet of milk and animal food, turpentine, possibly creasote inhalations, these are the most rational means.

Treatment of the Pleurisy and of the Pneumothorax.—If the gangrenous process reaches the pleura, producing a purulent pleurisy, or pyopneumothorax, it will be necessary to hasten to practice the operation of paracentesis, which alone can save the patient. (Bucquoi.)

NOTE.—For the following interesting case, illustrative of the antiseptic treatment of this disease, and the happy results which sometimes follow these peculiarly modern methods in cases which would otherwise be incurable, I am indebted to the Medical Record, vol. xx. p. 418.—Trans.

Pleurisy on the Left Side, with Exudation, Pulmonary and Pleural Gangrene, Ichoræmia—Complete Cure after Two Operations of Thoracentesis.—In the *Gazzetta Medica*, Dr. Zannini relates the following case: The patient, a lady aged forty, and of good constitution, was attacked with pleurisy on September 20th. The attending physician regarded this as an ordinary cold, and the patient, after a few days' treatment, was allowed to go about. A week later she was forced to go to bed again, suffering from a cough which brought up nauseating sputa. Another practitioner, being called in, diagnosticated pleuritis, with effusion on the left side, and pulmonary gangrene. Under rational treatment the patient improved, but on the 28th of October, a change for the worse took place. The face was bathed with a cold sweat, the features were pale and troubled, and the wandering eyes, cyanotic lips, and panting respiration, intermingled with sighs, expressed the anxiety provoked by the feeling of suffocation. The pulse was 150, respiration 60, temperature 104. The left pleural cavity contained both gas and fluid. Thoracentesis was performed, and two litres of a foul-smelling, purulent liquid, intermingled with putrid shreds of fibrinous membrane, were removed. The canula was allowed to remain, and three times a day the pleural cavity was washed out with a two per cent. solution of carbolic acid. In addition to this, the patient was put under antiseptic and tonic treatment, alcohol, quinine, and cinchona being administered; vapor of turpentine inhalations were also advised. Under proper management a favorable progress was made. One day, however, during an injection of the solution, the lady was seized with alarming cough, accompanied by symptoms of suffocation, and the writer was astonished to see the injected fluid issue from the mouth, the gangrenous process having created a communication between the bronchi and the pleural cavity. A diminution of the quantity and the number of injections became necessary, on this account. Upon two occasions the urine exhibited the characteristic olive tinge due to absorption into the system of carbolic acid, hence a hyposulphite solution was substituted. On November 16th, thoracentesis was again performed, the patient having developed symptoms of septicæmia. More than half a litre of a greenish matter, intermingled with gangrenous shreds of lung, issued forth, emitting an intolerable stench. Antiseptic injections, the first one of which was attended by a vomiting of the solution, were again practised, and finally the fluid returned as clear as it

entered. Subsequently the cavity became obliterated, and the patient recovered her health completely by the middle of May. The appetite and complexion are now good, the lady is well nourished, and not troubled by catarrh, cough, pain, or dyspnœa.

In the Gaz. Med. de Paris for Aug. 30th, 1885, is an interesting communication by Dr. Constantin Paul on the cure of Pulmonary Gangrene by inhalations from an ordinary inhaling bottle of phenic acid; the long tube being made to penetrate a stratum of water saturated with the acid. Dr. Paul reports seven cases successfully treated by this method. The patient is required to make the inhalations frequently, and ten minutes at a time, and to persevere with them night as well as day. These antiseptic inhalations are believed to prevent extension of the putrefying process and restrain the action of bacteria. The seven cases reported by Dr. Paul were examples of gangrene consecutive to embolism.—Trans.

SECOND SERIES.

CHRONIC SPECIFIC DISEASES.

NOTE.—This second series, which was written with the cö-operation of my clinical chief, Doctor Talamon, comprehends the specific diseases with chronic march, viz., syphilis, cancer and verminous affections.

PART V.

CHAPTER XXXVIII.

SYPHILIS OF THE LUNGS.

Delimitation of the Disease.

It is still difficult to-day to give a didactic description of syphilis of the lung. Although facts are not wanting, the notion of definite syphilitic alterations of the pulmonary parenchyma or of the respiratory passages has not been so readily accepted as that of the cerebro-spinal or hepatic lesions of the same nature. Syphilis determines on the skin and on the mucous membranes certain manifestations which are characteristic of themselves; a similar objective specificity has been asserted for the internal or visceral lesions. Unfortunately such a similarity does not exist. If it has been possible to find in the disposition of the cerebro-spinal or hepatic alterations certain characters peculiar to syphilis, these must needs be constant, and with respect to the lungs such distinctive alterations remain still to be found. Nay more, the notion of specific lesions must in the actual state of things be abandoned, as impossible to hold its ground before the new acquisitions of pathology. The lesions are always appreciably the same with acute evolution or with chronic march: the cause alone is specific. This may be illustrated by certain morbid changes which are special and which have enabled us down to the present time to give a definite denomination to the disease; for instance typhoid fever is characterized by intestinal ulceration; scarlatina by its angina and its cutaneous eruption; but between the lesions, visceral, hepatic, splenic, renal, etc., provoked by these two diseases there is no difference appreciable to the naked eye or to the microscope.

It is the same with respect to histology, and the morbid anatomist is unable to establish distinctive characters between the products of tuberculosis or syphilis. Embryonal proliferation and granular fatty degeneration, such are the near consequences of the pathological process engendered by the microbes in contact with the elements of the organism; sclerosis or caseification, such are the ultimate consequences. Only one thing, then, is specific: it does not pertain to the organism but to the outer world; it is not the elementary lesion but the morbid figured germ. If this germ is found, described, recognized, and recognizable, all becomes simple, in the case of necrosis as well as in that of tuberculosis. If the microbe be unknown, as it still is with respect to syphilis, all remains obscure or doubtful. Hence arises the opposition of certain medical authorities to the introduction of pulmonary syphilis into the specific pathological nosology.

Anatomical Characteristics.

In default of this specific parasitic element which still escapes us, it has been necessary to search for other criteria of the syphilitic lesion. The gumma has been considered a syphilitic product, admitted without dispute and apparently well defined; it is the gumma which has been arbitrarily invested with this attribute. In fact, when we consider the differences of opinion of the pathological anatomists with respect to the other pulmonary lesions observed in the syphilitic, one might be led to conclude that the gumma is the only alteration of the lung really syphilitic. But if a voluminous isolated gumma offers an aspect almost characteristic, it is no longer the same when the lesion is disseminated under the form of little nodules; there is not then any perceptible difference between these gummy nodules and tuberculous nodules. If one has recourse to the help of the microscope, the differences are completely obliterated; the structure is the same, and Cornil is of the opinion that it is very difficult to make the anatomical diagnosis of gumma of the lung from various tuberculous productions.

Gummy lesions or sclerous lesions; such are, according to Fournier, the lesions produced by syphilis in the lung as in the liver, the testicle, or the brain. But it is still more difficult to distinguish a syphilitic sclerosis from a sclerosis of other nature, than it is to distinguish a gummy syphilitic production from one of tuberculosis or of glanders. Tuberculosis often ends—perhaps as often as syphilis—in pulmonary cirrhosis. Certain simple inflammatory lesions of the pleura or of the bronchi may also determine a fibroid thickening of the connective tissue frame-work of the lungs; the different kinds of chronic lung affections due to inhalation of dust (pneumono-koniosis) are also characterized by pulmonary sclerosis. It is indeed possible to distinguish this latter by the nature of the different kinds of dust, the causes of the disease; but how are you to distinguish the other species? According to Virchow, there is a complete resemblance between the products of peri-bronchitis and of common chronic broncho-pneumonia and of the gummata or the sclerous broncho-pneumonia of the syphilitic.

Till, then, the specific microbe shall have been discovered, it will not be possible to obtain from the microscope a precise and certain criterion of the syphilitic nature of a pulmonary lesion.

In order to trace a more or less complete picture of syphilis of the lungs, it is to other elements that we must direct our attention. It is by the evolution, the mode of distribution, the microscopical aspect, the clinical march of the lesion, that one may venture to characterize it. And from this point of view it is convenient to make and keep distinct two great divisions which we cannot yet quite match the one to the other; the first being devoted to the study of the anatomical characters, the second to the description of the clinical character of the affection.

Anatomo-pathological Characters.

Pulmonary Syphilis of New-born Babes.—There exists a first variety of pulmonary lesions which belongs to hereditary syphilis and which offers but an anatomo-pathological interest; we shall be brief on this point. These lesions have been described by Depaul, Charles Robin and Virchow. They are met in fœtuses that have died before full term,

or were born dead, or in infants that have lived only a few days. They may present themselves under two aspects; sometimes under the form of disseminated nodules, which are hard, grayish, or similar to apoplectic infarctions, and to little metastatic abscesses; sometimes under the form of a diffuse induration, which Virchow has called *white pneumonia*, or white *hepatization*. The pulmonary tissue is hard, resistant to pressure and to section, creaking under the scalpel, and of a white color. Histologically the lesion is a diffuse interstitial pneumonia, very vascular, beginning in the peri-bronchial and peri-vascular connective tissue, then invading the inter-alveolar spaces, and gradually obliterating the pulmonary alveoli. The alterations of the alveolar epithelium are often very pronounced; you observe an epithelial proliferation so stuffing the alveoli, that Robin gave to this lesion the name of epithelial induration or epithelioma of the lung.

Syphilitic Pulmonary Lesions that are Indisputable.

I shall divide into two classes the alterations found in the lungs at the autopsy of adult syphilitics. The first class comprehends lesions whose syphilitic nature cannot be doubted; in the second category are ranged the alterations whose specific origin is probable but not yet absolutely demonstrated.

Among the genuine cases are those characterized by a syphilitic production in the form of a sclero-gummatous infiltration of the thoracic walls, developed primitively in the costal periosteum, or in the muscles, secondarily invading first the pleura, then the lung, penetrating the parenchyma to a greater or less depth. (Birkett.) Such cases are undisputed examples of syphilitic pulmonary lesions; they offer chiefly a pathoanatomical interest, for they enable one to study with certainty the alterations which the syphilitic processes may develop in the tissue of the lungs.

A second variety no less authentic is furnished by the voluminous gummata, completely circumscribed, which present in the lungs the special and well defined characters which they offer in the other organs.

In fine, facts which have been related where perfectly defined and recognizable syphilitic lesions of the larynx and of the trachea have extended along the bronchi and their finest divisions and thus reached the acini and the inter-alveolar connective tissue, seem to us to form a third variety, whose specificity is not less real than that of the two first groups.

Pulmonary Gummata.

The gummata of the lungs differ in nothing from those of other organs, and generally they coexist with productions of the same nature in some other point of the economy; a coexistence which, in doubtful cases, ought to be sought with care. These gummata are roundish masses, with more or less regular contours. They have a variable volume, from that of a pea or cherry to the size of a hen's egg or an orange. Small and scattered through the lung, they may give occasion for dispute as to their nature and be confounded with nodules of caseified tubercle. When voluminous they are quite characteristic, and the macroscopic aspect of the tumor suffices to declare its syphilitic nature. The morbid mass is consistent, compact, of a grayish color, almost translucent; at the period of crudity presenting a yellowish white tint; at an age a little more advanced

the tissue is friable, dry, and gives no juice on section. Each gumma is enveloped by a grayish fibrous zone which forms for it a sort of thick and resistant shell. Ordinarily few in number, but rarely isolated, you will generally find four or five of these gummata disseminated in both lungs. Often the lesions are limited to a single lung. As for their localization in the different lobes, no precise rule can be given. They have been found as well in the inferior lobe as in the middle or superior portions. All that can be affirmed is that they have no preference for the apex of the lungs.

Appearing thus under the form of voluminous masses which are compact and limited, the gummata are quite easy of recognition. But it is not the same when they begin to disintegrate. The gumma in fact, in the lung as in the skin or mucous membrane, undergoes little by little a granulo-fatty degeneration, which ends finally in a more or less complete ramollissement. Two consequences may result from this breaking down; a cavity or a cicatrix. The softening of the gumma indeed generally leads to ulceration of a neighboring bronchus, and gradually, sometimes indeed very rapidly, the gummy products are evacuated by expectoration. There remains then an excavation, generally of little size, but very much like a tuberculous cavity. "The walls of this cavity," says Fournier, "are lined internally by a white caseous substance, vestige of the gummatous tissue in process of elimination. Externally they are lined by a hard, condensed, grayish, fibroid tissue, a sort of external shell of gummatous neo-formation. This cavern, with its fibrous shell, may remain in this condition, or else the walls may come together, unite, and nothing is noted at the autopsy but a depressed and wrinkled cicatrix, the last vestige of the eliminated gumma."

It is apparent, then, that if the gumma of large size offers in the crude state characters sufficiently plain, when softened and in process of elimination it tends to become confounded with tuberculous excavation, especially in the forms of tuberculosis which end in sclerosis. The confusion is still greater when the gummata are small, not larger, for instance, than a pea or a cherry. Fournier has essayed to differentiate by the following characters the gumma of tubercle:

1. **The Situation.**—The tubercle is seated at the apex of the lung especially, and in both lungs; the gumma exists only in one side, as a general thing, and may localize itself in a portion only of the pulmonary tissue.

2. **The Number.**—The gummata are in general few in number; the contrary is true with respect to tubercles.

3. **The Color.**—The gummata are always white or yellow, never transparent like miliary tubercles.

4. **The Consistence.**—When it is not softened the gumma is harder than tubercles, and even when softened, it is still more resistant than the latter, on account of its fibrous shell.

Characteristics of this kind, which are true in a general and systematic sense, cannot evidently be of great utility in the doubtful cases where the tumors occupy both lungs, and the apices as well as the other parts, where their number is considerable or their volume and their consistency that of tubercle granula.

We have already said that the microscopical characters are absolutely insufficient to warrant a diagnosis. Histologically, in fact, how is the syphilitic gumma constituted? It was formerly believed that it was formed of a tissue similar to inflammatory tissue (granulation tissue), that is to

say, of round cells heaped together, and pressing against each other. A more close analysis has shown that the gummy lesions correspond to the same type of histological structure as the tuberculous lesions. To the latter had been successively given, as characteristic of it, first the giant-cell then the elementary follicle. The microscope has shown that the giant-cells, as well as the primitive follicle, exist in the syphilitic gumma. The syphilitic nodules or follicles of Malassez and Brissaud are in all respects similar to the elementary tuberculous granulations. They are like so many little inflammatory foci, provoked by the injection of irritating particles. Malassez admits two varieties of them: the lymphoid nodules formed by a mass of little round cells, and the epitheloid nodules, consisting of large granular cells. The two varieties are often united. When the nodules are agglomerated and fused together, especially when it is a case of lymphoid nodules, the distinction of component elements is almost impossible. The formation of these growths entails on the one hand the development of an interstitial inflammation of the frame-work of the organ, and on the other, the lesions of periarteritis and periphlebitis, which bring about a more or less marked narrowing of the calibre of these vessels. When the gumma is of long standing and has undergone caseous degeneration, you will, according to Malassez, observe three concentric layers: in the centre a fibroid tissue in a state of granular degeneration, presenting little bodies which are strongly refractive, taking a red stain with purpurine; in the middle portion a zone of fibrous tissue arranged in concentric layers with giant cells; and lastly at the periphery a zone of embryonal cells, invading the inter-alveolar spaces and the walls of the alveoli.

Nothing in this description is peculiar to syphilis, and the same dispositions are met in the tuberculous nodule. We have to-day, it is true, in Koch's bacillus a sure means of determining a tuberculous lesion. But we must not forget that in old lesions, and in particular in sclerous tubercles, the bacillus is very hard to find and may even be wanting, the parasite having been eliminated and leaving behind it only cicatricial modifications. This reservation being made, it is not the less incumbent on the clinician, when in presence of a case of fibro-caseous lesion of the lungs in a syphilitic individual, to consider the absence of the tubercle bacilli as an argument of great weight in favor of the syphilitic nature of these lesions.

Laryngo-tracheal and Pulmonary Lesions.

In this group there exist coincidently lesions of the larynx or trachea, and pulmonary lesions. The laryngo-tracheal lesions have a characteristic aspect; the pulmonary alterations do not differ from those which are observed in sub-acute or chronic broncho-pneumonia with sclerous induration. Certain authorities ask if the latter are not common non-specific lesions coinciding simply with specific lesions of the larynx. It appears nevertheless rational, when in the course of a laryngo-tracheitis, clearly recognized as specific, one sees develop the signs of a chronic inflammation of the bronchi or lungs, it seems, we say, rational, to attribute all these accidents to the same cause as is constantly done in the case of tuberculosis. However this may be, the laryngo-tracheal lesions present themselves under the three aspects of gummata, ulcerations and cicatrices.

The gummata are rare; they may form circumscribed or infiltrated deposits; their seat of predilection is the supra-glottidean portion of the

larynx; they may be seen there with the laryngoscope under the form of yellowish, firm, always quite voluminous prominences. The cases in which gummata have been noticed in the trachea or bronchi are sufficiently numerous; little indurated tumors of yellowish color have been observed by Wilkes and Moissenet in the sub-mucous tissue of the trachea, and Wagner says that he has found in the substance of the bronchial walls, little gummatous nodules. The most common lesions are ulcerations and cicatrices.

The syphilitic ulcer of the larynx is generally irregular, serpiginous, sometimes of considerable extent. The borders of the ulcer are sharply cut, and remain limited by red inflammatory areola. The bottom of the ulcer is grayish, of woolly, often of fungoid aspect. At the onset it is single, and rarely are there more than three or four, while tuberculous ulcerations are multiple from the very first; on the other hand, while laryngo-tuberculosis commences by the inter-arytenoidean mucous membrane, syphilis descends from the pharynx to the vocal cords, attacking first the epiglottis or the epiglottidian folds, before ulcerating the inferior vocal cords.

Certain lesions of the cartilages coexist habitually with these ulcerations, sometimes primitive, oftener consecutive to the process of ulceration; you note a peri-chondritis with ossification, necrosis and detachment of more or less extensive portions of the laryngeal cartilages. The process is the same as that which leads to destruction of cartilages in general and especially those of the nose. There may be perforation of the œsophagus, or abscess formation in the mediastinum.

The ulcerations of the trachea present the same characters. They are few in number, but quite extensive, with borders sharply cut, of irregular form, often invading the whole circumference of that air tube. They have their seat preferably in the inferior half of the trachea, on the level of the bifurcation (twelve times out of twenty-two cases reported by Gerhardt). In these cases they extend to the bronchi under the form of isolated or confluent ulcerations. When they are seated in the upper part of the trachea, they are simply the continuation of the laryngeal ulcerations. They may invade the entire length of the trachea. As in the larynx, these ulcers gain rapidly in depth, determining chondritis and necrosis of the cartilaginous rings. According to Vierling, in the majority of cases these tracheo-bronchial lesions are associated with similar lesions of the pharynx; this association has been found in thirty out of forty-six observations.

The cicatrices are the result of the healing of these vast ulcerations. Whether these cicatrices occupy the larynx, the trachea or the origin of the bronchi, the gravity of such a mode of healing is easily comprehended. The consequence can only be the narrowing or obliteration of a more or less extensive part of the respiratory passage.

In the larynx the cicatricial lesions present divers aspects. Sometimes fibrous bands, adhesions, more or less complete, unite and solder the inferior vocal cords. Sometimes there is retraction in mass of the entire cavity of the larynx; the cicatricial tissue draws the walls of the larynx towards itself, causing marked puckering and deformity, and the calibre of the tube is gradually reduced to a mere chink. In other cases the tissue of the cicatrix hypertrophies and forms an obstacle by itself to the passage of the air, or else upon the cicatrix, fleshy vegetations form after the manner of papillomata, and obliterate the air pipe.

It is the same in the trachea; projecting fibrous bands form veritable columnæ on the internal surface of the respiratory tube. By their retraction these bands diminish on the one hand the calibre of the canal, and on the other its length. The larynx is drawn down by this shortening; and when the tracheal cartilages have been destroyed, the air passage may be narrowed to the diameter of a goose quill. Similar cicatricial strangulations have been observed in the large bronchi.

As for the pulmonary lesions which sometimes accompany these ulcerations, or these tracheo-bronchial deformities, they have nothing characteristic, as we have said, but their coexistence with lesions whose syphilitic nature is notorious. They differ in nothing from the isolated pulmonary alterations which we are about to describe and which are still more or less a subject of dispute.

Pulmonary Lesions whose Syphilitic Nature is Still Under Dispute.

These lesions comprehend the fibroid phthisis of the English authors, Corrigan's cirrhosis of the lung, broncho-pulmonary sclerosis with all its consequences, chronic bronchial catarrh with or without dilatation of the bronchi, fibrous induration of the interstitial web-work of the parenchyma, sclerous thickening with firmly resistant adhesions of the pleura;—such are the alterations which are met with in certain forms of pulmonary disease, concerning whose origin there is much obscurity. Fibroid phthisis forms a complex group whose etiology seems to be multiple; it may be the result of inhalation of mineral dust (pneumono-koniosis, phthisis of miners, of stone cutters, knife grinders, etc.); it may be a mode of evolution of certain broncho-pneumonias called simple inflammatory; it may be the termination of certain tuberculoses with a peculiar march. We are now concerned to know if this complication may not also result from the alteration of the lung by the syphilitic poison.

Can syphilis determine in the pulmonary tissue lesions similar to cirrhosis of the lungs?

Is pulmonary cirrhosis frequently associated with visceral syphilis?

Do there exist special characters attributable to syphilitic pulmonary cirrhosis?

Such are the principal particulars to elucidate. The first point is established on the one hand by the existence of the white interstitial pneumonia of new-born babes affected with hereditary syphilis, which interstitial pneumonia is nothing but diffuse sclerosis; on the other hand, by the facts already cited above, that of Birkett for instance, where a syphilitic lesion of the costal walls gradually invades the neighboring pleura and lungs, producing a fibroid pleurisy and an undoubted interstitial pneumonia; syphilis may then determine the lesions of pulmonary sclerosis.

A statistical inquiry of Goodhart responds to the second question. Out of a hundred and seventy-seven autopsies of patients dying of visceral syphilis, autopsies made in the space of twenty years at Guy's Hospital, in forty-two the lungs were diseased. In six of these cases the lesions were simply those of acute pneumonia; the thirty-six other cases were chronic lesions. Out of this number, twenty-four were examples of fibroid phthisis; twelve were cases of ordinary tuberculosis. Hence one may conclude with Goodhart that when the lung is invaded in visceral syphilis, the lesions which it presents are generally sclerous in their character.

This is also the result of eighty-seven autopsies of pulmonary syphilis performed by Hiller.[1] But if the frequency of the alterations noted eliminates the idea of coincidence, one may still demand if the cirrhotic inflammation results directly from the syphilis or if it is not an affair simply of phlegmasic or even tuberculous lesions, which take on, under the general influence of syphilis, a sclerous character?

This brings us back to the inquiry whether syphilitic pulmonary sclerosis offers any special characteristics in its development or in its topographical disposition, which enable us to differentiate it from the other pulmonary scleroses.

Now on this point we are not yet thoroughly enlightened. Histologically, whatever may be the first cause, sclerosis is always like itself. According to some authorities, syphilitic sclerosis always begins around the arterioles. But besides the fact that there is nothing about the mode of onset peculiar to syphilis, the tissue of sclerosis forms muff-like investments quite as thick around the bronchi as around the arteries, and you will generally find certain air tubes and blood-vessels lost and, as it were, choked out in the midst of a dense tissue of compact fibroid lamellæ studded with flat cells ramified and radiating, and with a few embryonal elements. This same fibroid tissue infiltrates the inter-alveolar spaces, the sub-pleural tissue, constricting the alveoli, which contain a granular detritus with a few nuclei. In fact there do not exist, according to Goodhart, any microscopic differences between syphilitic cirrhosis and the sclerosis of tuberculosis, of anthracosis, or of simple pneumonia. The same conclusion results from researches made by Colomiatti.

The macroscopic disposition of the lesions offers characters of greater value. Depressed and radiating cicatrices, nodosities scattered along the bronchi, a marked thickening of the pleura, belong especially to syphilitic sclerosis. The two observations of Pawlinoff and of Lancereaux may be considered as types of the kind. The pleura forms a fibrinous shell, thicker than a centimetre in places. The lungs are, as it were, ploughed with deep furrows, filled with scelerous tissue. These furrows, resembling syphilitic cicatrices of the liver, deform the surface of the lung, determining more or less marked retraction of the parenchyma. In other places the cicatrices produce, in the very interior of the lung, white bands which seem to radiate from a common centre. Between these tracts the parenchyma is indurated or emphysematous. Sometimes an entire portion of a lobe is transformed into a fibrous block, grayish in color, creaking under the scalpel. In fine the nodosities are perceptible on palpation of the lung, appearing at the surface or disseminated in the depths of the parenchyma; they are yellowish, dry, rounded, from the size of a pea to that of a walnut, presenting a central part which is caseified or formed entirely of fibrous tissue. They are seated in the neighborhood of, and sometimes even in the walls of the bronchial tubes. If you add to this, that the bronchi are ordinarily dilated, presenting sometimes here and there sacciform or ampullar dilatations, you will have an aggregate of lesions which are almost characteristic. We should add that these lesions are generally bi-lateral; nineteen times out of twenty in Goodhart's cases.. It may happen, as will easily be understood, that the alterations are the less pronounced and the less generalized the more the nodosities are wanting, and that all may be limited to a circumscribed thickening of the pleura,

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[1] Charité Annalen, t. ix., 1884.

and to fibrous bands partitioning off a portion of the lung. In such cases the aspect is less demonstrative than in the cases of extensive sclero-gummatous lesions; it is then that the discovery of similar lesions in the liver, in the testicles, furnishes important data for the anatomical diagnosis. In such instances, moreover, the sclerosis rarely occupies the apices. It invades preferably the base of the lungs, and especially the root of the large bronchi, extending to the circumference of the hilum as from a centre of radiation.

To sum up, one may observe in the lung true syphilitic gummata having the same characters as the gummata of any other organ. New-born babes present a form of interstitial pneumonia which is indisputably syphilitic. In adults syphilis may also produce a pulmonary cirrhosis which may be either circumscribed or diffuse. When diffuse it represents one of the varieties of the phthisis called fibroid, and offers an aggregate of macroscopic characters which united may enable us to recognize their origin, although the microscope may be incapable of furnishing any differential sign. Sometimes, however, the macroscopical characters are too incomplete to warrant an absolute diagnosis. It will be necessary then to take especially into account the coexistence of tracheo-laryngeal lesions or of sclero-gummatous lesions discovered in other organs, liver, kidneys, testicles, bones, etc., without forgetting that pulmonary tuberculosis may develop in a syphilitic patient as well as in any other subject.

CHAPTER XXXIX.

CLINICAL CHARACTERS.

As syphilis may determine in the lungs destructive and ulcerous lesions on the one hand, and sclerous lesions on the other, it results from this that there does not exist a single sign which is pathognomonic of pulmonary syphilis. The physical signs in fact are those of the one or the other of these two processes, the functional symptoms are those of every alteration of the bronchi or lungs: signs of induration or of softening, symptoms pertaining to cough, dyspnœa, muco-purulent or hæmorrhagic expectoration, which belong to syphilis of the lung as well as to tuberculosis, and even to cancer. At the same time clinically pulmonary syphilis has an existence, being attested especially by the cases of recovery obtained by means of the specific treatment, of which the real value, without being absolute, is undeniable here as in every other lesion supposed to be syphilitic. It is in cases of pulmonary gumma that we oftenest witness curative results, and although the assertions of Pancritius with respect to the frequency of these alterations are without doubt exaggerated, and although we may not know with certainty, when the disease ends in recovery, what variety of anatomical modifications the affected parenchyma has undergone, it is reasonable to admit the existence of a gumma, or if one prefers to employ the expression of Moncry, of a specific plastic infiltration susceptible of resorption.

But by the side of these facts of recovery, there exist cases were the specific treatment remains almost if not quite impotent, and which are not the less surely cases of syphilis.

Such are the pulmonary lesions which coincide with laryngeal or tracheal lesions clearly syphilitic, and lastly, as final term of the series, syphilitic fibroid phthisis, where the organization of the sclerous tissue is too advanced to offer any chances of resorption under the influence of any treatment whatever. We admit, then, that pulmonary syphilis may present itself under three clinical aspects:

1. The gummy infiltration, circumscribed or more or less diffuse.
2. Laryngo-tracheo pulmonary syphilis.
3. Pulmonary sclerosis with or without bronchiectasis.

Signs of Gummatous Infiltration.

It is impossible as yet to exactly superimpose the clinical signs on the anatomical lesions of pulmonary syphilis. The word gummy infiltration does not then apply absolutely to the alterations which we have previously described under the name of pulmonary gumma; it indicates simply the probable lesion of the lung in individuals who having been treated by mercury and iodide of potassium have got well, while it was at the same time impossible to tell precisely what were the anatomical characters of this pulmonary lesion. The functional symptoms are those which in every

affection of the bronchi or lungs attract the attention towards the respiratory apparatus; the cough, the oppression, a muco-purulent expectoration and hæmoptyses.

Neither the cough nor the dyspnœa offer anything special. It has been said that hæmoptysis is rare; others have maintained that it is only less common than in tuberculosis. Pulmonary syphilis being itself a rare affection in comparison with tuberculous phthisis, it seems to us very difficult to establish a relation between the frequency of hæmoptysis in these two diseases. What is certain is that spitting of blood has been mentioned as a prominent symptom in a great number of cases of syphilitic lesions of the lungs. As, on the other hand, tuberculosis may undergo evolution without provoking the least bronchial hæmorrhage, one cannot make either of the absence or the presence of this symptom a differential characteristic.

As for the expectoration, it is muco-purulent or purulent, but thus far, as in the case of tuberculous sputa, no peculiarity has been noted which can aid in the diagnosis. The fact of Cabe is probably unique. In this remarkable case at the moment when the gummy infiltration was softening and undergoing disintegration, the patient coughed up sputa containing yellowish lumps the size of a large bean, which together weighed twenty grammes. The microscope showed that these lumps were formed of the débris of pulmonary tissue, in which were recognized alveoli stuffed with epithelial cells and granular detritus and surrounded with brilliant fibrous zones; these sclerosed zones contained in places heaps of embryonal cells, and one could distinguish also among them the débris of blood-vessels and of bronchioles with thickened walls. This peculiar expectoration coincided with the appearance of signs of softening and of pulmonary excavation, lesions which got well in two months under the influence of mercurial frictions. The evolution of the gummy infiltration comprehends in fact two periods; a period of induration and a period of ulceration with formation of cavities. As for the physical signs, the dullness, the absence of vesicular murmur at first, then the blowing respiration or tubular souffle afterwards, characterize the first period; the excavation signs, bubbling râles, cavernous breathing and pectoriloquy reveal the second stage. According to most authorities, the most important characteristic is furnished by the localization of these signs.

Gummy infiltration is said almost always to occupy the middle portion of the lung and more especially still, the right median lobe. According to Grandidier, out of thirty cases of curable pulmonary syphilis, in twenty-seven this special localization existed; once only the left lung was affected; twice the morbid signs were noted at the right apex. It is, then, in the interscapular region that we should look for the physical signs of pulmonary gummata. The souffle has its maximum on a level with the hilum, losing itself insensibly laterally, above and below; the apex and the base are free from morbid sounds. But although Tiffany and Pancritius maintain the same view, it is far from being true that this special localization is constant. In a great number of clinical facts published by other writers, the physical signs existed as well on the left side as on the right, as well at the base or apex as in the middle portion, and in particular, in the remarkable case communicated by Fournier to the Academy of Medicine, not only was the lesion seated on the left side but it was limited to the apex of the lung. On the other hand, in all the observations followed by autopsy, there is nothing to confirm the assertion

of Grandidier and Pancritius; the gummata occupied the most diverse parts of the lungs, and according to the statistics of Goodhart, pulmonary syphilis is oftenest bilateral; "nineteen times out of twenty-four."

The General Phenomena are Variable.

From this point of view Fournier distinguishes three categories of facts; cases in which the lesion remains absolutely latent; cases characterized only by functional troubles, and local physical signs, the constitution not suffering; cases, finally, where grave general symptoms are associated with the local signs. In fact the existence of constitutional phenomena depends on the extent and profundity of the lesion. If the gummy infiltration is limited, if it continues in the state of induration without tending to ulcerous softening, it is compatible with a state of fair general health, and Bazin undoubtedly makes allusion to facts of this kind when he says that individuals affected with syphilitic phthisis, go about as persons in good health. They have little cough and expectoration and but slight dyspnœa. But when the gumma softens and destructive and extensive lesions are produced, the general appearance of the patient is that of a tuberculous subject in the second or third stage. The emaciation, the general loss of strength, the cachectic yellowish tint, the hectic fever with evening exacerbations and abundant night sweats, the anorexia and diarrhœa are as marked then as in ordinary phthisis, and death may supervene with all the symptoms of tuberculous cachexia.

Signs of Laryngo-tracheo Pulmonary Syphilis.

This second variety is essentially characterized by the signs of ulcerous laryngitis or of tracheal stenosis, due to the ulcerations and cicatrices which we have described above. When these alterations are very pronounced they dominate the situation, and it is almost impossible to decide if the lung is also in its turn affected; one finds himself in the same conditions as in presence of tuberculous laryngeal phthisis with pulmonary lesions of little extent. In other cases, on the contrary, the symptoms on the part of the lungs attract attention; the clinical attendant is inclined to attribute them to a commencing tuberculosis, when the existence of the characteristic lesion of the larynx or epiglottis comes to clear up the diagnosis.

Functionally, the symptoms of laryngeal and tracheal syphilis are confounded, being those of every grave laryngitis. The ulcerations manifest themselves by modifications of the voice, which progress from hoarseness to absolute aphonia, by cough, pain on pressure, dyspnœa more or less marked, wheezing, with laryngeal whistling. When the cicatricial tissue tends to contract and obliterate the air tube, the dyspnœa becomes extreme, attacks of suffocation set in; it is a tug for breath, with asphyxia imminent, and demanding surgical interference and tracheotomy.

The existence of troubles of the voice and aphonia, is the only sign which enables one to diagnosticate tracheal stenosis pure from laryngeal or laryngo-tracheal stenosis.

As for the physical signs, they are furnished by the laryngoscopical examination; they are not characteristic, nor have they any importance, except at the period of gummy infiltration, or at the period of ulceration. The gummata present themselves under the aspect of tumors which are voluminous in comparison with the tubercle granule, varying from the size

of a pea to that of a walnut, yellowish and protruding from beneath the mucous membrane. The ulcers are few in number, irregular, with indurated borders, surrounded by an inflammatory zone. They are seated at first on the epiglottis and its folds, and have a descending march, contrarily to what is the case in tuberculous ulcerations. In fine they co-exist ordinarily with ulcerous lesions of the same nature in the pharynx.

Signs of Pulmonary Sclerosis, with or without Bronchiectasis.

Whether you have to do with syphilitic sclerosis or any other kind of sclerosis of the lungs, the subjective, objective, and general signs are the same. It seems, then, useless to give a symptomatological description of this kind, a description which belongs more properly to the general history of fibroid phthisis. It is to this form that ought to be attributed, without doubt, all cases of pulmonary syphilis with slow march, with relative conservation of the general health, characterized by a chronic catarrh with intermittent acute or sub-acute exacerbations, and presenting to auscultation signs of bronchial dilatation or of chronic induration, more or less disseminated through the parenchyma. It is impossible to find either in the evolution or in the physical signs, the least argument in favor of the syphilitic nature of the sclerosis. Here also, however, the lesions appear to descend along the bronchial tubes and to present, in consequence, the maximum of the stethoscopic signs on the level of the tracheal bifurcation. But this characteristic, useful perhaps at the onset, has no longer any value when the alterations are already of a certain age and have consequently invaded a considerable extent of the lungs. The sclerosis being recognized, the question of diagnosis limits itself to a search for the cause.

Diagnosis.

To diagnosticate a syphilitic alteration of the lungs, appears to many physicians an inexcusable temerity. To reassure these timorous spirits, it suffices to remark that twenty years ago clinicians used to regard likewise, as medical curiosities, without any practical value, the few cases published as examples of cerebral syphilis. Who would dare to deny to-day the frequency of syphilitic lesions of the cerebro-spinal axis and the importance of their diagnosis? While, therefore, recognizing, and having always present to mind, the possibility of simple coincidences, and while freely admitting that the frequency of pulmonary tuberculosis is such that in presence of a chronic affection of the lungs the idea of tuberculous lesions ought to first suggest itself in preference to any other diagnosis, it remains none the less certain that the pulmonary tract remains open to other parasites as well as to the tubercle microbe. But of all the parasitic affections with progressive march, syphilis is, next to tuberculosis, the most common; while this should not lead one to say that all that is not tuberculous is syphilitic, it should nevertheless be at least a reason why one should not admit too readily the idea of pure coincidence, when one finds himself in presence of chronic pulmonary alterations, not of a tuberculous nature, in a syphilitic individual. Light, then, towards a correct diagnosis can be obtained from a knowledge of the antecedents. We must, moreover, take into consideration, as diagnostic factors:—

1. The existence of previous and especially of present syphilitic lesions; as pulmonary syphilis always belongs to the third period of the infection,

it is tertiary accidents which should be sought for, such as cutaneous gummata, pharyngeal and laryngeal ulcerations, tumor of the testicle, cerebral manifestations, etc.

2. The localization of the lesion elsewhere than at the summits, and in particular at the level of the hilum, especially on the right side, the right median lobe.

3. The rapidly curative influence of the specific treatment by mercury and iodide of potassium.

4. Finally in cases where the diagnosis hesitates between tuberculosis and syphilis, the absence, as proved by repeated examinations, of Koch's bacillus in the sputa.

When it is a case of limited gummy infiltration, the localization of the signs of pulmonary induration on the level of the right median lobe in an individual unmistakably syphilitic according to his own confession, or presenting some other plain tertiary manifestation, such as brachial monoplegia, for example, or a syphilitic sarcocele, may be held as a characteristic of great value. If these signs disappear rapidly under the action of a mixed treatment, or treatment by the iodide alone, the diagnosis may be regarded as certain.

If the gummy infiltration occupies the apices and tends to take on an invading march, as in the observation of Fournier, determining hæmoptysis, fever, and all the signs of general cachexia, the diagnosis will present no great difficulty, except so far as the case may be confounded with tuberculosis; it must be either the latter or syphilis. Then, however, the existence of tertiary lesions in different parts of the body will not suffice to remove all the doubts by reason of the seat of the gumma in the upper parts of the lung. It is then that the histological examination of the sputa is absolutely demonstrative; the absence of Koch's bacillus might justify one in ruling out tuberculosis. The detection of the bacillus need not, however, exclude all notion of pulmonary syphilis, as it is possible for syphilis and tuberculosis to co-exist in the same subject and in the same organ.

It is also with tuberculosis, having its seat in the larynx, that laryngo-pulmonary syphilis offers points of resemblance. Here also the discovery of Koch's bacillus in the sputa or in the mucosities removed by a swab from the surface of the ulcerated parts, would decide the question. The laryngoscopic examination and the march of the lesions present also certain differences. We have said that the seat, the number, and the aspect of the ulcerations are not absolutely the same. In syphilis it is especially the epiglottis and its folds which are the most profoundly affected; in tuberculosis it is the interarytenoidean mucous membrane. The syphilitic lesion follows a descending march from the pharynx and epiglottis towards the bronchi; tuberculosis, on the contrary, ascends from the glottidean regions towards the pharynx.

As for the diagnosis of pulmonary sclerosis, the question comes back to this: pulmonary sclerosis, complicated or not with bronchial dilatation, being observed in a syphilitic patient are you authorized in referring the pulmonary lesion to the infection of syphilis? If there exists no *professional*[1] cause for cirrhosis of the lungs, if the phenomena of bronchial catarrh do not go back to childhood, and only date from a few years, if the lesions seem to predominate about the median parts, if the syphilis

[1] As in the anthracosis of coal miners, etc.—Tr.

has previously made certain marked and deep inroads on other organs, if there exist signs of hepatic or cerebral syphilis, we should be strongly inclined to admit the syphilitic origin of the pulmonary sclerosis; but we are assured that till new light is obtained from further discoveries, no certain or irrefragable proof of this specific origin can be clinically furnished.

Prognosis and Treatment.

The prognosis is relatively benign in the case of gummy infiltration, on condition, however, that the nature of the lesion be recognized, and the proper treatment be early instituted. Even at the period of softening and excavation, and of general cachexia, complete cure is possible by the mixed treatment. As for laryngo-tracheal syphilis which has become ulcerous, or retractile and sclerous, the prognosis is always very grave. If one succeeds by an appropriate medication in bringing about the cicatrization of the ulcers, the stenosis of the larynx and of the trachea will not be less to be dreaded. Tracheotomy alone can remedy the disasters of laryngeal stenosis; as for tracheal stenosis, despite tracheotomy, death by progressive asphyxia or by suffocation is the rule. The consequences of pulmonary sclerosis are less disastrous; cirrhosis of the lung is assuredly compatible with life for a time, which may be very long. The patient is not the less incessantly exposed to bronchial or bronchio-pulmonary complications, and ends by succumbing to the marasmus proper to chronic affections of the respiratory tree. It is none the less necessary in these different cases to administer with perseverance iodide of potassium in large doses, sixty, seventy-five, ninety grains a day; it is, in fact, doubly indicated both on account of its specific action on the sclerous products of tertiary syphilis, and its special action on the broncho-pulmonary manifestations with dyspnœic or asthmatic tendencies.

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Note.—In the *New York Medical Journal* (August 1st, 1885) Dr. William H. Porter contributes a paper—"Phthisis and Pneumonia in their Relation to Syphilis." His observations are founded upon a study of one hundred cases in which these affections were associated, and we draw a resumé of them worthy of the careful attention of our readers.

He defines pulmonary syphilis as a condition of the lungs in which there is a progressive thickening of the walls of the vesicles; this change is often associated

with inflammatory deposits, gummy formations, which, degenerated into cheesy masses, become encapsuled or liquefy, and give rise to cavities.

The first necropsy, in connection with a typical clinical history, was made in 1878, and in this instance there was no question of the existence of acquired syphilis, and the exemption from inherited phthisis was equally positive. Since that time a number of cases have been noted.

Of the one hundred patients chosen as the basis of Dr. Porter's paper, fifty-two were males and forty-eight females, which he says does not represent the relative frequency among patients at large, for the whole number treated showed it to be more frequent among females.

"The rational signs in many respects resemble those of a tubercular phthisis. The patients complain of having had a heavy cold with incomplete recovery, followed by a dry, hacking cough, or one with an abundant muco-purulent expectoration. The sputum is either white and frothy or thick, purulent and of a greenish or yellow tinge, as in true pulmonary tuberculosis. Early hemorrhages are frequent and often copious, while the dyspnœa is an early and pronounced symptom. Pleuritic pains are common, and there is often great weakness, while the general physique remains fairly good—in fact the emaciation is not at all in proportion to the weakness, and is nothing like that of the tubercular variety. The skin is warm and moist, but there is little or no elevation of the bodily temperature. Night-sweats are frequent and distressing, and the patient complains of cephalalgia and of indefinite and wandering pains in the bones and tissues, which increase at night. Dyspepsia and slight jaundice are not infrequent. The urine has a characteristic pale, limpid appearance, which, together with the sallow complexion, often leads to the suspicion of a nephritic lesion. The urinary examination is usually negative unless there happens to be a coincident renal lesion.

"Married females abort without any apparent exciting cause further than the syphilitic taint. Their children, if born, are often plump, but soon die of marasmus. The first child is often healthy, but the subsequent children are delicate. Frequently patients complain of sore throat and of an early loss of hair.

"The physical signs are peculiar and diagnostic; the respiratory act is labored and all the accessory muscles of respiration are brought into play, but there is little or no expansion of the chest. Early in the disease palpation reveals increased fremitus, but in the advanced cases it is diminished, owing to the small volume of air entering the lung.

"Percussion gives varying degrees of dullness, which is most marked at the apex, but diminishes toward the base. The percussion-note is dull and of 'wooden quality,' and localized areas of dullness are found. Owing to the fact that small cavities have thin walls and contain considerable air, the dullness is not so great as would be expected, but, compared with a sound chest, it is decided.

"Auscultation yields most positive and peculiar evidence when the rational history is taken into consideration. The inspiratory and expiratory murmurs are prolonged and harsh, especially the former, with a decided intermission between the two. Broncho-vesicular breathing is frequent at the apex. When the amphoric whisper is absent the presence of a cavity is eliminated. As a rule, crepitant and subcrepitant râles are absent, thus eliminating chronic bronchitis. It is differentiated from asthma by the absence of the sibilant and sonorous sounds. The vocal resonance is exaggerated over the consolidated portions. Another very strong and pathognomonic sign is a peculiar pain and œdema of the sternum and of the tibial crests. Pressure over these regions produces a very peculiar pain, which is quite intense and accompanied by a recoil not easily forgotten when once recognized. Patients often try to avoid giving evidence of this, but as surely fail, as though trying to resist the muscular reaction to the electrical current. A noticeable feature is that when the sternum is excessively sensitive the tibial crests are less so, and *vice versa*. There is no other disease in which this localized tenderness in œdema of the periosteum is met with. It is never found accompanying a true miliary tuberculosis, but it is exceedingly common in connection with syphilis, and, from this absence in cases of tuberculosis where the diagnosis was confirmed by the necropsy and its almost universal presence in cases of syphilis, it may be regarded as pathognomonic of a specific taint. In addition to this, it is always found to yield when the patient is under an anti-syphilitic régime.

"The diagnosis is based upon five principal signs and symptoms:

"1. The abundant expectoration without any signs of softening of the pulmonary tissue.

"2. The weak and debilitated condition without marked emaciation, and the good rational history of phthisis.

"3. Pronounced dyspnœa without any evidence of a cardiac or pulmonary obstruction to the circulation.

"4. The peculiar pain and the reaction to pressure upon the sternum and tibial crests.

"5. The ready response to treatment is another element in the diagnosis.

"The prognosis depends upon the early recognition and treatment. Syphilitic phthisis may run a very rapid course, but, as a rule, it is quite chronic. Dissipation hastens its progress, especially when alcoholic stimulants are used to excess.

"In treating this affection the liver should first be freely acted upon by cathartic doses of calomel or podophyllin, which in some cases should be frequently repeated. Experience has shown that the mixed treatment yielded better results than when mercury or iodide of potassium alone was used. The combination most successful is one-sixteenth of a grain of the biniodide of mercury, from five to ten grains of the iodide of ammonium, and ten to twenty grains of the iodide of potassium in a drachm of the compound tincture of gentian, three times a day after meals.

"Great reliance is placed on the ammonium salt, not perhaps on account of its intrinsic action in syphilis, but from the action which it has in rendering the two others more soluble, capable of easier absorption and assimilation, and in increasing their solvent action upon the newly formed gummatous elements—in fact, its action may be compared to that of trituration on calomel.

"The conclusions may be summed up as follows:

"1. *Ætiology.*—Pulmonary lesions attributable to syphilis are very common, more so in females than in males, with the maximum number of cases occurring between thirty and forty years of age; it is as frequently, if not more frequently inherited than acquired.

"2. *Pathology.*—Is most frequent at the apex; usually involves both lungs; is a peculiar pneumonic process in the early stages, while later cavities are formed, and it becomes phthisical in the sense of progressive consolidation, followed by softening and the formation of cavities. There is a strong resemblance, but a positive difference, between syphilitic and tubercular phthisis, and a positive anatomical difference between a syphilitic and a miliary tubercle.

"3. *Symptoms.*—These are peculiar and diagnostic.

"4. *Diagnosis.*—This rests mainly upon the rational history and physical signs, the extreme dyspnœa, the periosteal tenderness and the absence of an increased bodily temperature.

"5. *Prognosis.*—This depends upon an early recognition of the trouble.

"6. *Treatment.*—It must be anti-syphilitic to be of any avail. Many cases are unaffected by iodide of potassium alone, unless under enormous doses, but a rapid improvement follows upon the use of the biniodide of mercury, iodide of ammonium and the iodide of potassium."

The foregoing observations and similar studies by the same author (*Boston Medical and Surgical Journal*, April 23, 1885), deserve at this time careful study, for we believe that much of the lung disease which has passed for phthisis among syphilitic negroes, probably accords with Dr. Porter's description of syphilitic pneumonia more than phthisis. Here is a wide field for post-mortem examination which could be cultivated to great advantage by those physicians whose practice is largely among the negroes. Persistent effort would probably, in time, overcome their superstitious opposition to post-mortem examinations. At any rate, in the public hospitals of the South there must be sufficient material for the purpose.—North Carolina Medical Journal, Aug. 1885.—Tr.

PART VI.

CHAPTER XL.

CANCER OF THE LUNGS.

Mode of Development.

We shall have to include in one and the same description, cancer of the lungs, cancer of the pleura, and cancer of the mediastinum. Clinically it is impossible to make a distinction between these three lesions. From the point of view of prognosis, the gravity and the consequences are the same. In fine, from an anatomo-pathological point of view, the three organs are ordinarily simultaneously invaded, and if the alterations predominate sometimes in this or that part, one cannot draw any conclusion of practical import from this fact. It is, then, useless to multiply divisions which can have no serious foundation. As much may be said respecting the anatomical varieties of cancer. Whether the carcinomatous growth be encephaloid, scirrhus, melanotic, etc., the symptoms are the same, and aside perhaps from certain differences in the march of the disease, nothing warrants us in making a separate study of each of these varieties.

In fine, there exists a primary cancer and a secondary cancer of the lungs; but here also, as well from the point of view of the clinical manifestations as from that of the aspect of the lesions, the two modes of cancer must be comprehended in the same description.

It is, however, not without advantage to enumerate the divers pathoanatomical species of cancer which are met with in the lungs, and to indicate the differential characters which they offer in this organ as in every other part of the economy.

Histological Varieties.

The kind of cancer ordinarily observed in the lungs is encephaloid, a term applicable rather to the external macroscopical appearances than to any well defined histological structure. It is generally carcinoma that takes on this aspect, but other tumors may present the same appearances, sarcoma and epithelioma for instance. We do not purpose here to discuss the histological nature of carcinoma of the lungs. It is well known that two views are prevalent; the one that of Virchow, of Cornil, and Ranvier, derives carcinoma from the connective tissues; the other, which Robin has always maintained, which Waldeyer has adopted, and Malassez has accepted with certain modifications, regards cancer as an epithelioma.[1] This last

[1] "Cancer is a disease with hypergenesis and divers cellular alterations of the epithelial system, especially of the deep epithelia, *i.e.*, of the glandular or non-glandular parenchyma oftener than of the teguments. No error is greater than to consider cancer as an affection having its starting-point in the cellular tissue,

view tends to be triumphant everywhere at the present day, and as far as it concerns the lungs, Malassez, by his remarkable analysis of a case of encephaloid cancer which nobody would ever have hesitated to regard as an alveolar carcinoma, has shown that the epithelioma which he calls *metatypical*, may develop at the expense of the walls of the pulmonary alveoli. The primary lesion consists in an epithelial formation on the internal surface of the alveoli; the cells of new formation have some of them normal or adult cylindrical or cubical forms, these are the *typical* cells; others are of varied forms which are irregular and abnormal, and are the *metatypical* cells in process of development and proliferation, and about to invade the connective tissue and lymphatic spaces. They constitute the true infecting cancer, and the larger part of alveolar carcinomata are nothing but metatypical epitheliomata. However this may be, to the naked eye, the encephaloid presents itself under the aspect of a milky white mass, closely resembling the white substance of the brain, offering in places a rosy hue; this mass is of a softish consistency, sometimes almost diffluent, giving, when cut, an opaque milky liquid—the cancer juice.

The scirrhous variety, which under the microscope is characterized simply by a more considerable thickening of the fibrous framework of the carcinoma, forms masses which are harder, and are disposed in patches, or nodules of lardaceous aspect. Melanotic cancer is characterized by its black aspect, its sepia colored juice which oozes on section; it is ordinarily a sarcoma, but carcinoma may also present this same infiltration of cells with black pigment. Melanotic cancer is generally consecutive to the ablation of a tumor of the eye of the same nature.

In colloid cancer the tumor has the appearance of a gelatinous mass, infiltrating the lung more or less extensively. This mucous transformation belongs chiefly to the carcinoma variety.

These are the four principal varieties of cancer said to occur primarily in the lungs. But all the other malignant tumors, whether carcinomatous, epithelial, chondromatous, or osteoid, may develop with their proper characters in the pulmonary tissue.

Topographical Varieties.

In the lungs as well as in the liver, primary cancer is said, as a rule, to assume the massive form (*en masse*), while secondary cancer takes on the disseminated form, being diffused through the parenchyma as distinct nodules. This is true in a general way. But on the one hand, you may observe a single voluminous mass occupying the lung and the mediastinum coincidently with the existence in a neighboring viscus or in some part of the periphery of the body, of a tumor of the same kind which may be of quite small size; and, on the other hand, the primary cancer may give origin to secondary nodules either in the lung which it first invaded, or in the opposite lung.

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or other tissues of mesodermic origin. All the lesions of these tissues are only consecutive to their invasion or erosion by epithelial productions proliferating more energetically than they, and encroaching upon them by reason of atrophy of the walls of the tubes of the parenchyma, and of the hyaline substratum of the tegumentary membranes. This fact explains the penetration of epithelial cells into the lymphatics of the affected organ, where they continue to multiply by segmentation, filling these vessels and mapping them out as by an injection, sometimes even in regions far removed from the organ which was the point of departure. The cells, nevertheless, preserve there the characters of those in the original tumor."—Charles Robin, in Nouveau Dictionnaire de Médecine.—Tr.

It will not do then to make too much account of this division from an etiological point of view. But the two topographical varieties of cancer in mass or cancer in disseminated nodules are sometimes observed at the autopsy combined, sometimes only one of them is present.

What one most generally meets with, is a voluminous indurated mass, involving the roots of the lungs, occupying the ganglia and the cellular tissue of the mediastinum, and penetrating to a greater or less distance into the pulmonary parenchyma along the large bronchial tubes. The œsophagus, the trachea, the large veins at the root of the neck, the phrenic and pneumogastric nerves may be compressed, altered, choked out in the morbid mass; this is the mediastinal form of pulmonary cancer, which seems to begin in the peri-tracheal and peri-bronchial glands or in the connective tissue of the mediastinum.

In another variety of this massive form, the pulmonary parenchyma itself is destroyed and transformed by the neoplasm. It is an entire lobe, the entire half of a lung, which constitutes the cancerous mass; the other lung may be sound, however, but it generally contains numerous nodules distributed through its substance. The mediastinal glands are also altered and invaded in this form; but are not fused into a mass quite as dense and compact as in the first variety.

Finally, in that form of cancer characterized by disseminated nodules, tumors, variable in number and volume, are seen throughout the whole extent of the lung. This form is almost always bilateral. The lung may be riddled by these cancerous nodules; at other times only six or seven of them are met with in the different lobes. The nodules are ordinarily sub-pleural, but they may exist also in the substance of the parenchyma.

It is to this form especially that belongs the kind called sub-pleural cancerous angioleucitis, which maps itself out on the surface of the lung under the aspect of streaks, networks, or irregular polygons, which are nothing but superficial lymphatic vessels injected with the cancerous matter. In cases of cancer of the abdomen or of the breast, infecting the lymphatic system, it may happen also that the nodules shall occupy only the serous surface, and that not a single nodule may develop in the parenchyma of the lungs. Generally, however, in pulmonary cancer the pleura is affected either with adhesive inflammation, or with an inflammation of a fibrino-serous or hæmorrhagic nature.

CHAPTER XLI.

SEMEIOLOGY, DIAGNOSIS AND TREATMENT.

Cancer may invade the pulmonary tissue and this even to a considerable extent, without anything revealing its existence or even engaging the physician to make a careful physical examination. Verneuil and Potain have related cases where more than half of a lung was destroyed by cancerous masses without the attention of any one being directed to the chest.

Walshe has cited the case of an individual affected with melancholia who died in a state of extreme cachexia, without ever have presented the least pulmonary symptom such as cough, dyspnœa, or expectoration.[1]

The autopsy showed both lungs studded with cancerous nodules. In this observation, which is of exceeding interest, the cancer was primary. Ordinarily these examples of latent cancer belong to the secondary forms; surgeons are especially apt to notice them at the autopsy of patients operated upon for tumors of the breast, of the bones, of the subcutaneous tissue, etc., in which event neoplastic masses more or less numerous are found disseminated through the lungs, while no signs during life may have indicated their presence. But such cases are rare, and the localization of cancer in the lungs manifests itself generally by the signs common to every broncho-pulmonary affection, that is to say, by cough, oppression, and thoracic pains.

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[1] [Walshe's observation, to which reference is here made, concerned a patient suffering from melancholia, and is thus related by him: "About six months previous to admission into the hospital on December 21, 1858, he became subject to failure of memory and fits of rage, sometimes threatening to murder his wife. Took to his bed three weeks since from bodily weakness; has since been drowsy and stupid, but has hardly slept at all. Whilst in the hospital he lay on his back with a dull expressionless countenance. He was with difficulty got to answer any question, and then only in a low tone and with extreme slowness; it was elicited that he had slight headache, but no pain of any description elsewhere. He had no paralysis, no rigidity of any of the muscles. He lay still in bed, asking for nothing and passing his motions and urine under him, but, as it seemed, merely from disinclination to move. After being in the hospital about a week he began to refuse food, which, however, was forced down him, with wine, bark, etc. He gradually became weaker and died on January 11, 1859, apparently from inanition. No other symptoms occurred before death, except that the breathing had become rather labored *during the last few hours*. He was quite conscious up to the last, and had no paralysis, and no impairment of the senses; he had resisted receiving his medicine shortly before death, endeavoring to push the glass away with his hands, and after the medicine had been got down, his face put on an expression of disgust with the taste. He had no cough, nor other symptoms directing attention to the chest. Post-mortem: the right lung infiltrated throughout its lower half with encephaloid cancer; the left lung crowded with nodules of the same, from a pin's head to a pea in size. Brain presented numerous rounded cancerous masses studded throughout its whole substance; these varied in size from a small pea to a large nut; there were certainly more than twenty of them." — Trans.]

Local Symptoms of True Pulmonary Cancer.

The *cough* offers nothing special in cancer which is exactly limited to the lung; it is dry or soft, like the cough of ordinary tracheo-bronchitis. But when the mediastinal glands are invaded it may present a spasmodic, whooping-cough character, which in default of other signs should awaken the attention to the possibility of a compression of the trachea or pneumogastric nerves. The cough then becomes paroxysmal, extremely painful, with noisy inspiration determining symptoms of congestion, with cyanosis of the face and lips and sometimes vomiting of food. It will not do, however, to attach to a cough of this kind a special importance, for it exists with the same characters in pulmonary tuberculosis and in most cases of tracheo-bronchial compression, whatever may be the cause.

The *dyspnœa* is rarely wanting. It is permanent, becoming aggravated under the influence of the least effort, as brisk walking; at other times it presents nocturnal exacerbations resembling cardiac asthma. The compression of a bronchus or of a vagus nerve augments still more its intensity. In the case of cancer in mass, involving the roots of the lungs the dyspnœa equals and even exceeds the orthopnœa of cardiac asystolia. It reminds one of the continuous distressed breathing of patients suffering from a vast pericardial effusion. In mitral asystolia, the oppression results especially from the passive congestion of the lungs, from the venous stasis, which prevents the renewal and oxygenation of the blood through contact with the air. In cancer of the lung, the cause of the dyspnœa is not only the impediment to the circulation, but there are other adjuvant causes. Not only the circulation in the pulmonary arteries and veins is embarrassed, but the penetration of the air is hindered in consequence of the stenosis of the bronchi or trachea, and to these two mechanical obstacles may be joined the compression of the pneumogastric or recurrents. Hence it is that the patients, seated on the edge of their beds and arched forward upon their hands, with tumefied face and neck, protruding eyes, livid lips, straining with all their might to compel a little air to enter their chest, offer a pitiable spectacle of respiratory anguish.

This intense dyspnœa belongs, however, only to the advanced periods of the disease. It is not so with the pain, which from the very first stages may be of exceptional severity. According to Walshe, the pain is wanting in secondary cancer. This is true when you have to do with the disseminated nodular form; but in the massive form, secondary or primitive, the pain is almost constant. It may be dull and obscure, but even then is remarkable, by its localization in one side, by its persistency and its tendency to become steadily worse in spite of all treatment. At other times it is acute and lancinating, resembling intercostal neuralgia, or even the pains of vertebral caries or cancer. In a case of Olivier, it was accompanied by an intercostal zona. In certain cases also there have been observed painful irradiations into the shoulder and even down the arm of the corresponding side; this neuralgia of the brachial plexus may be even the first symptom of the disease, as in the observation of Behier

Currant-jelly Expectoration and Engorgement of the Infra-clavicular Glands.

Two signs have been given which are almost pathognomonic of pulmonary cancer; these are the currant-jelly sputa, and the engorgement

of the infra-clavicular glands. It was Stokes and Marshall Hughes who first pointed out the value of the first of these signs; Behier has especially insisted upon the second. The symptomatic importance of these signs has been contested; the objection has been made that they are not found in all cases of cancer of the lungs and that they may be observed in other affections. The first objection has no great significance; it is evident that these signs have no value except on condition of their existing. Stokes and Behier admit this, and have not pretended that their absence proves the non-existence of cancer. As for the second objection, it reduces the difficulty to a question of diagnosis; other pulmonary lesions may give rise to a bloody expectoration and an engorgement of the infra-clavicular glands, but does this bloody expectoration correspond to what Stokes and Hughes have described under the name of currant-jelly sputum? Does this glandular engorgement present the characters of the cancerous engorgement described by Behier? These, then, are the characters which it is important to decide.

If one contents himself with saying that an engorgement of the glands of the infra-clavicular hollow indicates cancer of the lungs, the proposition thus enunciated cannot be sustained; for this engorgement may exist in adenitis, in tuberculosis, and even under the influence of a simple inflammation. The engorgement, to have the value attributed to it, ought to present its proper characteristic:

The glands ought to be voluminous;

They ought to offer special hardness without resistance or elasticity;

They ought to be indolent.

The woody induration differentiates them from the engorgements of tuberculosis or of adenitis. Their large size and the absence of pain distinguish them from inflammatory engorgement. Thus particularized, this sign in an individual affected with a chronic pulmonary disease, has a value almost pathognomonic.

We may say as much of the currant-jelly sputa. "I am disposed," says Marshall Hughes, "to regard the expectoration of a viscous rosy-colored liquid, of an aspect quite peculiar, as of great value in diagnosis." Stokes is a little more precise. The sputa by their color and their consistency resemble currant-jelly; they have not the viscosity of pneumonic sputa. But he does not regard this expectoration as characteristic of pulmonary cancer except when associated with the following signs: a continuous pain in the chest, a varicose state of the veins of the heart, thorax, and abdomen, an œdema of a member, the rapid formation of external cancerous tumors. In reality the veritable currant-jelly sputum ought to have the following characters:

It is of a gelatinous consistence, forming in the spittoon a softish trembling mass which is neither viscous nor adherent like the pneumonic sputum;

It is of a rosy color and not of a rusty or brick-dust appearance;

It is homogenous, almost transparent, like a well-made jelly.

It does not, therefore, resemble either the sputum of pneumonia or that of pulmonary apoplexy.

It is the more important to well differentiate it from the two varieties of bloody expectoration which may be observed in cancer of the lungs.

Hæmoptysis itself, spitting of pure blood, is quite frequent, and in an observation published by Liouville, repeated hæmoptyses each day occurred for three months, presenting sometimes the characters of ordinary

tuberculous hæmoptysis, sometimes the aspect of hæmoptœic sputa, sometimes the distinctive appearance of gooseberry jelly.

A real cancerous expectoration has been observed in a certain number of cases. Thus it is that in an observation of Aviolat, the sputa had a milky white color which was due to a mixture of bronchial mucus with particles of encephaloid matter. Hyde Salter observed also in a patient of his, sputa of a thick compact consistency sinking to the bottom of the spit-cup, and formed of little distinct masses resembling particles of cooked veal; unfortunately the microscopical examination of these particles was not made. In a case of melanic cancer reported by Lancereaux, the sputa contained little blackish masses similar to tobacco seeds; the microscope showed that these blackish grains were constituted by heaps of cells filled with granulations of black pigment. Finally, Walshe says that he has discovered in the expectoration of several patients real cancer cells; but here the diagnosis is more delicate, for the cancer cell has nothing about it absolutely characteristic.

Hæmorrhagic Pleurisy.

A sero-sanguinolent effusion into the pleura has been regarded also as pathognomonic of pulmonary cancer. It is Trousseau who has especially contributed to give prominence to this relation (which he has, however, made much too absolute), between the hæmorrhagic inflammations of serous membranes and cancer. The sanguinolent exudation exists in cancer of the lungs, but the fibrino-serous exudation is quite as frequent. On the other hand, without taking into account the general diseases which are attended with hæmorrhage, many other pulmonary lesions may determine a hæmorrhagic pleurisy, such as miliary tuberculosis, and especially chronic inflammation of the pleura, pachy-pleuritis, due ordinarily to alcoholism. So, considered by itself, sero-sanguinolent effusion into the pleura has no value from the point of view of the diagnosis of pulmonary cancer. It does not acquire the semeiological importance which Trousseau attributed to it, except in the case of an individual suffering from obscure and ill-defined symptoms, which one would naturally refer to an intra-thoracic tumor, or to chronic pneumonia, where one succeeds by puncture of the pleura in withdrawing a bloody liquid; in these conditions, due account being made of the antecedent manifestations, the symptom becomes a powerful argument in favor of a lesion of cancerous nature.

Symptoms of Compression.

Quite a new series of symptoms may result from displacement or compression of neighboring organs by pulmonary cancer. These symptoms, it is easily understood, are not observed in the case of disseminated nodules, unless the glands of the mediastinum are simultaneously affected. They belong especially to massive cancer of the lungs, or to mediastino-pulmonary cancer.

The compression may involve all the organs contained in the mediastinum. Those which are the most ordinarily affected are in order of frequency:

(1.) *The superior vena cava and its branches,* brachio-cephalic trunk, subclavian and jugular veins. The compression of these veins, the frequency of which is explained by the very slight resistance of their walls, gives rise

to two signs quite ordinarily observed in cancer of the lung. These signs, the inevitable consequence of every kind of prolonged obstruction to the venous circulation, are dilatation of the superficial venous net-work, and œdema of the upper part of the body. The venous dilatation manifests itself by the exaggerated development of the supra-clavicular thoracic and even the abdominal venous plexuses; this turgescence of the venous canals, which may present themselves under the aspect of bluish flexuous cords, may be limited to one side of the thorax. It is the same with the œdema, which at the beginning is always unilateral and generally localized at first in certain regions. Hence it is, also, that one may observe in the first place a swelling limited to one side of the face and neck, or perhaps an œdema of a single muscular group. The œdema rapidly increases, invades the whole of one side of the body, then all the supra-diaphragmatic regions; it may even become generalized, constituting total anasarca.

(2.) *The Trachea and Recurrents.*—Hoarseness of the voice, aphonia, wheezing and dyspnœa, first paroxysmal, then continuous, reaching such an intensity that in one case, recorded by Valcourt, it was believed that there was a foreign body obstructing the air passages, and tracheotomy was performed;—such are the signs which appertain to compression of the laryngeal nerves and larger air tubes. These signs, joined to the œdema of the upper portions, and dilatation of the thoracic veins, are scarcely ever wanting in mediastino-pulmonary cancer.

(3.) *One of the Subclavian Arteries.*—The compression of one of the subclavian arteries, a rare event, produces an inequality in the two radial pulses, an habitual symptom of aneurism of the aorta.

(4.) *The Œsophagus.*—The dysphagia which results from compression of the œsophagus is rarely complete. There is a very marked impediment to deglutition, with a sensation of arrest of the alimentary bolus behind the sternum; sometimes even there are regurgitations; a congeries of phenomena which may deceive the medical attendant into the belief that he has to deal with a case of cancerous stenosis of the œsophagus.

(5.) Finally, we must mention the possibility of compression of one of the sympathetic nerves, with inequality of the pupils, etc., or of the branches of the pulmonary artery, with dilatation of the right heart; the aorta even may be pressed upon; but such compressions have been exceptionally noted. What is more frequent, is the displacement in mass of the heart in the cases of massive cancer of the left lung. Lateral detrusion of the heart may ensue from pressure to the right, and this organ may beat under the right nipple, as in cases of vast pleural effusion. It is possible also that the cancer may develop as an extended sheet, formed at the expense of the sub-sternal cellular tissue lining the anterior thoracic parietes in front of the heart, like a neoplastic cuirass. The heart is in such cases forced backwards; its apex beat can no longer be detected; the pulsations appear to auscultation as if muffled and distant, and by reason of the extent and situation of the dullness as well as of the dyspnœa, which is extreme, one might be led to believe that there was a vast pericardial effusion (as in the case of Laboulbène).

Physical Signs.

Laennec, who has given but a very vague description of pulmonary cancer, contents himself with saying that "the stethoscope will indicate the existence of encephaloid growths in the lungs when they form volumi-

nous masses, which is common enough in this species of accidental production." "There are," says Stokes, "no physical signs peculiar to cancer of the lungs. Pulmonary cancer can only give rise to signs of induration or of compression. Now such signs appertain to many other lesions besides cancer. The clinician, however, observes on inspection, auscultation, and especially on percussion of the thorax, certain signs which we will now indicate.

Inspection.—Inspection may not disclose any very marked modification of the thoracic cage. At other times one may observe either a certain ampliation of the affected side, or much oftener, according to Walshe, who denies absolutely all ampliation, a localized retraction with immobility of the side. In any event the most important phenomena, which should always be sought for when pulmonary cancer is suspected, are as follows: Dilatation of the superficial venous plexuses; Unilateral œdema of the thoracic parietes; The existence of indolent glands of a woody hardness in the supra-clavicular fossa.

It is, in fine, possible that the cancerous tumor may appear externally in a bulging part of the thorax, as in an observation of Heyfelder, where there was observed on the diseased side an indurated mass as large as the mamma of a female. There may even be developed multiple little tumors in different parts of the body, as in the subcutaneous cellular tissue.

Palpation gives contradictory signs from which no certain datum can be derived to assist diagnosis, even supposing all idea of pleurisy excluded. With a cancer purely pulmonary one may in fact note a diminution and even a complete abolition of the thoracic vibrations.

Auscultation.—By itself pulmonary cancer gives rise to no morbid sound. When it is disseminated under the form of isolated nodules, of little size, the intervening pulmonary tissue remaining perfectly healthy, the respiratory sounds present from beginning to end their normal characters. When, on the contrary, the cancer forms a compact block in which all vestige of parenchyma has disappeared, it is the complete abolition of the vesicular murmur which is remarked. The silence is absolute, as in the case of a large pleural effusion, or in a massive pneumonia, and for the same reasons. But in fact it is very rarely so. Whether the tumor compress the bronchial tubes, whether the infiltration be incomplete, whether congestive and inflammatory phenomena ensue in the neighboring alveoli or bronchi, almost all the varieties of morbid stethoscopic bruits may be heard. Sometimes it is a tubular souffle, either bronchial or amphoric, hollow, sonorous, absolutely dry, without the accompaniment of the least râle; sometimes the bronchial souffle is surrounded by a more or less extensive zone of sub-crepitant râles, the index of foci of congestion or of peripheral œdema; sometimes you have the signs of a unilateral or double bronchitis, with snoring, sibilant or mucous râles, which predominate, and the respiration only takes on a blowing character on a level with the root of the bronchi. A bronchophonic resonance of the voice ordinarily co-exists with the bronchial or tubular character of the souffle. In other cases all symptoms of a subjacent lesion are masked by the signs of pleurisy with effusion.

The true excavation signs, cavernous souffle, gurgling and pectoriloquy, are always wanting in cancer of the lungs, and the persistence of the signs of induration without tendency to softening, is not one of the least important of auscultatory phenomena which have a bearing on the diagnosis of the lesion. But it is easy to see that one may be often deceived by the exis-

tence of the sub-crepitant râles of congestion or catarrhal inflammation in the neighborhood of the tumor and imagine the commencement of a softening which does not exist.

Percussion.—Percussion probably furnishes the physical signs most likely to awaken suspicion as to the existence of pulmonary cancer; not that these signs have anything characteristic in themselves; the dullness, with loss of elasticity, which is observed in cancer of the lung, belongs as well to pleurisy, to hydatid cysts, etc. But the delimitation of this dullness presents here, at least in certain cases, something special enough to excite the attention. It has no place of election, as in tuberculosis; it commences where the neoplasm commences, and develops with it. Often it occupies only one of the sides of the chest; the healthy pulmonary tissue, crowded towards the other side, gives a normal or tympanitic sound. When the dullness occupies the anterior part of the chest, on the right side or on the left, when it is seated in the sternal region, the posterior parts being sonorous, it is of great diagnostic value in connection with the functional symptoms before enumerated.[1]

General Symptoms and Clinical Aspects.

The general symptoms of cancer of the lung are rather those of a chronic affection of the air passages than the ordinary symptoms of the cancerous diathesis. Doubtless one may observe the straw-yellow color, the emaciation, the peripheral œdema with venous thrombosis, but the signs of the cancerous cachexia, properly so called, are rarely witnessed; the cough, the hæmoptysis, the repeated bronchites, the pleural complications coinciding with the wasting and the loss of the forces, recall rather the symptomatic aggregate of pulmonary tuberculosis. If to this are joined signs of compression, dyspnœa with paroxysmal suffocations, cyanosis, œdema, anasarca, the general aspect is then more or less like that of patients affected with cardiac dilatation consecutive to broncho-pulmonary sclerosis, but in nothing resembles that of a cancerous individual.

The evolution of pulmonary cancer may be absolutely latent, as we have before said. It may also be acute or chronic, the latter being the rule.

What is very strange is that the two abnormal forms, latent and acute, in almost all cases belong to secondary cancer. We shall not repeat what we have said respecting latent cancer. As for acute cancer, one may find at the autopsy either a sort of miliary carcinoma, under the form of nodules studding the two lungs or enormous masses of encephaloid tissue.[2] It is well known how rapidly encephaloid productions develop in certain cases. In an observation of Jaccoud, in eight or ten days an encephaloid cancer had filled the entire upper part of the anterior mediastinum, and had assimilated to itself the whole of the upper lobe of the right lung. Clinically the disease presents itself sometimes under the aspect of an acute pleurisy, this is the diagnosis which is oftenest made; sometimes under the form of a generalized bronchitis with excessive dyspnœa. In certain

[1] A good illustrative case is recorded in the Lancet., Dec. 13, 1884, p. 1047. There was enlargement of the right side with displacement of the liver; absolute dullness with abolition of vocal fremitus; currant-jelly expectoration, etc. There was also co-existent pleurisy with slight sero-sanguinolent effusion. There was bronchial breathing with crepitant râles over the dull area. The post mortem revealed the new growth filling the air cells and bronchi, and undergoing softening —Trans.

[2] See the instance recorded in the Lancet, loc. cit. This case is especially instructive, as being complicated with tuberculosis.—Trans.

cases the diagnosis of acute galloping phthisis has been made; in others the only symptoms have been a constantly increasing dyspnœa with a little cough, ending in six weeks in death by a progressive asphyxia. (Carswell.) The duration of this acute form varies from five or six weeks to eight or ten days.

We may admit three principal aspects of cancer with chronic march: a bronchial form; a pleuritic form; and a compressive form.

In the first form, the disease manifests itself for a variable time by repeated seizures of bronchitis similar to the acute catarrhal outbursts which attend tuberculous phthisis, or chronic catarrh of the air passages. In the course of these bronchial attacks, there appears a sanguinolent currant-jelly expectoration, or repeated hæmoptyses. A notable symptom is dyspnœa, with vague pains in the chest. The physical signs are those of chronic bronchitis with pulmonary sclerosis. A few indurated glands may be observed in the infra-clavicular fossa; the patient wastes and loses strength, has a sallow cachectic appearance—it is the marasmus of chronic diseases—and death comes in to end the scene.

In the *pleuritic* form, whether ushered in by the signs of bronchitis or an indefinable state of malaise and prostration, or whether the signs of pleurisy were the first phenomena noted, the pleural effusion dominates the situation. Sometimes the pleurisy begins with the symptoms of an acute frank inflammation, sometimes it develops insidiously. The respiratory embarrassment, which is always considerable, demands thoracentesis; this operation gives issue to a liquid which is often hæmorrhagic, which is also sometimes fibrino-serous. Thoracentesis, however, does not ordinarily give any relief, and the liquid is sure to rapidly reproduce itself. In one case the patient was tapped as many as twenty-five times, and at the autopsy the pleura contained still two quarts of sanguinolent liquid. The patient generally succumbs in this form to asphyxia, sometimes, however, to the extreme cachexia.

In the *compressive form* the phenomena of venous stasis and of dyspnœa predominate. In the course of a protracted dyspnœa, at first light, afterwards more severe, with now and then fits of suffocation, sometimes accompanied with hoarseness of the voice, aphonia, or wheezing, you will see sometimes produced an œdema localized in one side of the face, or neck, or in one of the superior members. This œdema rapidly increases. You notice at the same time a varicose dilatation of the veins of the infra-diaphragmatic regions. Percussion gives a wooden dullness occupying one of the regions of the anterior thoracic parietes, with abolition of the vesicular murmur in this point, or with production of a tubular blowing sound, which is dry, without râles, situated at the root of the bronchi.

The symptoms of venous stasis are aggravated, the œdema becomes general, sub-crepitant râles occupy both lungs; the cyanosis, the dyspnœa, the asphyxia attain an extreme degree, and the patient dies suffocated, or in a comatose state, due to ventricular paralysis by compression of the jugular veins, or superior vena cava.

Diagnosis.

Without designing to attribute to them any absolute value, we are warranted in saying that the most important signs relating to the diagnosis of cancer of the lungs are the following:

The special bloody expectoration, which is translucent, not viscid, having the color and consistency of currant jelly;

The existence of glands of a woody hardness, indolent and voluminous, in the supra-clavicular fossa;

The varicose dilatation of the veins of the thoracic walls and of the neck;

The unilateral œdema of the face or of an arm;

The existence of a cancerous tumor in some organ, or in some region of the body;

The traces of an old operation for the ablation of a tumor, whose most habitual seat is the mammary gland, the genital organs, or the eye;

The physical signs of an extensive induration of the lungs, persisting without symptoms of softening, coinciding with a very marked dyspnœa and an intra-thoracic pain rebellious to all treatment.

These signs when co-existing, give a certainty which is almost absolute. They should be carefully sought whenever pulmonary cancer is suspected.

Tuberculosis.—Having gone thus far in the diagnosis, with what affections may cancer of the lungs be confounded? The differentiation from pulmonary tuberculosis seems easy. It is difficult in any case to mistake a tuberculous for a cancerous patient, although the error has sometimes been committed; but it is possible in presence of cancer of the lungs to mistake it for tuberculous phthisis. The differential signs are nevertheless plain enough.

The signs of tuberculosis predominate in the apices and occupy both lungs. The signs of cancer always predominate on one side, and have no seat of predilection.

Even in total caseous infiltration of a lobe or of a whole lung, the only form of tuberculosis which can really be confounded with cancer, there are always signs of softening superadded to the signs of induration. The latter constantly exist alone in cancer during the entire duration of the disease.

The dullness of caseous pneumonia never presents the character of wooden resistance peculiar to cancerous dullness; it is never circumscribed like the latter.

The dyspnœa is easily supportable in tuberculosis, it is always intense in cancer. When there exist engorged supra-clavicular glands in phthisis they have neither the hardness nor the volume of cancerous glands. The currant-jelly sputa are never seen in tuberculous phthisis.

Finally, and this sign is pathognomonic, the absence or the frequence of the tubercle bacillus, if the diagnosis is between cancer and tuberculosis, will decide absolutely the nature of the disease.

Diagnosis from Sclerosis and Hydatids.

The diagnosis of cancer from pulmonary sclerosis presents great difficulties. The physical signs may be the same, being those of chronic bronchitis with more or less extensive induration of the lung; and it is known that in pulmonary cirrhosis, these signs, as in cancer, generally predominate in one side of the chest and without seat of predilection. If the cancer be primary, if it be exactly limited to the lung, if there exist no glandular induration, the diagnosis will be necessarily hesitating. In this case, the march of the disease may give some light. Pulmonary sclerosis is a lesion with very slow evolution, taking long years to develop. Cancer, on the

other hand, in its longest duration does not exceed two or three years. The diagnosis will be easy when the currant-jelly sputa appear, or when you note the production of supra-clavicular engorgement, or the signs of mediastinal compression. In such cases also the repeated histological examination of the sputa may perhaps reveal the presence of portions of cancerous débris. In any doubtful case the diagnosis of pulmonary cirrhosis is the only one admissible as long as you have not for guide any one of the characteristic signs which we have indicated above; but such an event would be rare.

Hydatids.—There is another affection of the lungs quite infrequent in our country, which may be mistaken for cancer. I refer to hydatid cysts in their first periods, especially when seated at the apex and in the anterior parts. The diagnosis is not a difficult matter. You base your opinion chiefly on the bulging, limited to the cyst; on the exact circumscription of the dullness, which can be mapped out with a pencil; on the sudden transition, which is noticeable to auscultation, between the absolute silence of the respiration and the existence of the normal vesicular murmur; in fine, on the duration of the disease, which is compatible for long years with a fair state of general health.

Diagnosis from Pleurisy, etc.

Practically the true difficulties concern the diagnosis of the pleuritic and compressive forms. In the first place, cancer of the lungs may exactly simulate pleurisy with effusion; in the second place, pleurisy is a frequent complication of cancer. It is especially in the massive cancers invading almost the whole of the lung with absolute dullness, abolition of the vocal vibration, and vesicular murmur, that the mistake will be committed, and there is really no means of obviating it except by thoracentesis. It is in such cases that the discovery of any of the signs enumerated above, such as dilatation of the thoracic veins, the supra-clavicular glands, a previous operation practised on the eye, the testicle, the mamma, etc., should awaken attention and inspire suspicion of cancer. When pleurisy exists, a delicate point is to determine if it be symptomatic of a subjacent cancer. It will not do to hasten to the conclusion that you have a cancerous tumor to deal with because a bloody fluid has been withdrawn from the pleura by tapping; we have already sufficiently insisted on this point; on the other hand, the existence of a serous effusion does not prove the absence of cancer. A dyspnœa out of proportion to the abundance of the exudation in a cachectic individual, the absence of any relief from this oppression after thoracentesis, the rapid reproduction of the liquid, are arguments in favor of the hypothesis of a pulmonary lesion marked by effusion. But if no other more characteristic sign is noticeable, neither the march of the pleurisy nor the characters of the liquid can furnish data sufficient to warrant one in predicating the nature of the pulmonary lesion.

Aneurism.—Two diseases may simulate mediastino-pulmonary cancer: aneurism of the arch of the aorta and adenitis. The physical signs are the same: retro-sternal dullness, signs of compression of the trachea or bronchi, of the recurrents, of the sympathetic, inequality of the radial pulse, etc. On the other hand, the dyspnœa, the cyanosis, the impediment to the supra-diaphragmatic circulation, etc., are the same in both cases.

It may, however, be affirmed that for the aortic aneurism to present a dullness as extensive as that of mediastinal cancer, it must attain con-

siderable dimensions, and thereupon the pathognomomic signs will be obvious enough, to wit, the pulsatile tumor with beatings and murmurs distinct from those of the heart. These stethoscopic signs will differentiate the aneurism even from cases where the cancer forms a tumor which is uplifted by the beatings of the artery, as in instances given by Stokes.

Adenitis.—As for adenitis, a single circumstance will aid you in the diagnosis from cancer. I allude to the existence of glandular tumors situated in other parts of the body, in the axilla, along the neck, in the abdomen. If the adenitis were localized in the mediastinal glands, the diagnosis would be impossible, and would offer besides but little interest, for it may be affirmed that there is an insensible transition from adenitis to carcinoma by lymphadenoma and lymphasarcoma.

Treatment.

Death being the fatal consequence of the disease, the rôle of the physician is restricted to moderating the cough, calming the pain, and assuaging the dyspnœa. In the pleuritic form it is useless to multiply punctures, when the liquid is so rapidly reproduced; and resort should be had to tapping only when the abundance of the exudation adds a new anguish to the other causes of dyspnœa which torture the patient.

PART VII.

CHAPTER XLII.

HYDATID CYSTS OF THE LUNGS.

Etiological Conditions.

The researches of Liebold, of Kuchenmeister, of Cobbold, have shown that the eggs of the tænia echinococcus, the habitual parasite of the intestine of the dog, introduced into the human organism, undergo development there under the form of hydatid cysts. It is well known that in our climate the ordinary habitat of the six-hooked embryo of this tænia is in the liver. The pulmonary localization, although possible, and observed in a certain number of cases, is quite exceptional. Hence it is that it is not from documents furnished by physicians of our country that the history of this affection can be written. We are obliged to consult the memoirs of foreign physicians, and in particular those of Iceland and Australia, for complete information respecting both the etiological conditions and the symptoms and prognosis of the disease. In France the treatise of Davaine supplies but few facts concerning the history of the echinococcus. The thesis of Hearn (1875), where have been grouped all the facts at that time published of hydatid cysts of the lungs, contains only 147 cases, and most of these are taken from the Australian journals. Australia is, in fact, together with Iceland, a country where hydatid cysts are frequently observed. According to Eschricht, the sixth part of the population is affected with this disease; at Victoria, in Australia, moreover, in the space of eleven years two hundred persons succumbed to the affection.

But if in a general way the frequency of hydatid cysts appears to be equal in the two countries, it is not so with the localization of the echinococcus. In Iceland, as in the rest of Europe, it is the liver which is the ordinary seat of the disease. Out of 255 cases, in fact, Finsen observed hydatids of the lung but seven times. In Australia, on the contrary, the lung is frequently stricken; out of 254 cases of hydatids, Thomas observed the disease in the liver in 183, and in the lungs in 71. On the other hand, in sixteen years, Dongan Bird had an opportunity to observe 150 cases of pulmonary cysts.

Fixation of the Tænia.

How does the embryo of the tænia come to obtain its habitat in the pulmonary tissue? This is a question still imperfectly solved. The routes pursued by the same embryo to gain access to the liver are themselves but ill determined, and if we admit as a rule that it is by the system of the mesenteric veins, and of the vena porta, that the echinococcus is carried to the hepatic tissue, the fact in reality has not been demonstrated.

Are we to believe that the parasite also follows the blood-courses when it finds its habitat in the lungs? This is possible in cases where the pulmonary cyst is secondary to a cyst previously developed in the liver; from the hepatic veins the embryo may easily be transported through the inferior vena cava, the right heart and the pulmonary artery, to the capillaries of the lungs, where it is arrested. But it is less easy to understand the possibility of this way of transportation when the pulmonary hydatid exists alone, for in this case we must conclude that the echinococcus, taking its departure from the surface of the intestine, traverses the hepatic capillaries, without being arrested there, despite the conditions which ordinarily determine such arrest.

Hence it is that it has been supposed with good reason that the eggs of the tænia penetrate the pulmonary tissue directly by the inspired air. Without doubt the kind of life led by miners and Australian farmers, their cohabitation with dogs, explains, as MacGillivray has shown, the frequency of hydatids in Australia, but these conditions cannot make intelligible the pulmonary localizations. Moreover, according to Dongan Bird, hydatid cysts are quite as common at Victoria among the rich as among the lower classes. It is, then, quite a probable supposition, as Bird suggests, that the excrements of the shepherd dogs, so numerous in this country, excrements containing in great quantity the ova of the echinococcus, when dried in the warm winds and sunshine, and pulverized by being trodden under foot in the streets, are wafted about by the atmosphere in the form of fine dust, and may thus penetrate the respiratory passages in the inhaled air.

To recapitulate, we know that the hydatid of the lungs, like the hydatid of the liver, comes from the tænia echinococcus of the dog; it is then sufficiently probable that cohabitation with the dog is one of the conditions the most favorable for the development of the disease in man, and this is in fact what observations in Australia as well as in Iceland demonstrate; but we are ignorant why the parasite localizes itself here in the liver and there in the lung; we are also ignorant by what channel it enters the lungs, although Bird's hypothesis of the direct inhalation of the germs through the air seems to be the most probable.

Lesions Produced by the Hydatid Becoming Encysted.

It is well known how the embryo of the tænia echinococcus comports itself when once arrested in the tissues. It there develops under the form of the *mother vesicle*, of gelatinous appearance, formed of numerous concentric layers. According to Huxley, the most internal layer alone ought to be considered as the embryonal vesicle; the external investments being only a product of the parasite's secretion. In this sack is found a clear limpid liquid, where float other vesicles sprung from the first, some of which remain adherent to the parent vesicle. The hydatid thus constituted may attain a volume which varies from the size of a cherry to that of the head of an adult; but whatever may be its size, it provokes around it an irritative process which ends in the formation of an envelop of connective tissue, *membrana adventitia*. This envelop, according to the age of the hydatid, may present a fibrous, cartilaginous, or even chalky appearance.

The fertile or germinative membrane, the anhistous membrane, and the membrana adventitia or connective tissue envelope; such are in fact the

three constituent investments of every hydatid cyst. We shall not here describe either the characters of the liquid contained in the sac or the divers aspects presented to the microscope by the scolex of the echinococcus with its free or invaginated head and its crown of hooks. These peculiarities differ in nothing from those which characterize the hydatid of the liver.

Davaine has, however, indicated a feature which has a great importance from the point of view of the precise determination of the place of development of intra-thoracic hydatids, namely, the absence of the adventitious membrane around vesicles developed primarily in the pleura. If this criterion be admitted, it will be seen that true hydatid cysts of the pleural cavity are very rare. In most of the observations of this kind published by writers, the case was one of hepatic cysts bulging through the thinned diaphragm into the pleural cavity, or of hydatids developed in the subpleural pulmonary tissue. In 150 observations, Bird saw but a single instance of pleural hydatid. In reality, although the echinococcus may fix itself and grow in the pleural cavity, and even in the cellular tissue of the mediastinum, such facts ought to be regarded as exceptional. It is in the tissue of the lung itself that the parasite ordinarily grafts itself.

One may, moreover, meet with the cyst in all parts of the lung, as well on the right as on the left side, in the upper lobe as well as in the inferior and middle lobes. Nevertheless it is more frequent in the right lung, and it oftener occupies the inferior than the superior lobe. The echinococcus may be solitary or multiple; a voluminous cyst may be found in one lung, and a number of small ones in the other. In fine, the hydatid may be exclusively pulmonary, or it may occur at the same time in several other organs, and in particular in the liver.

Here are the figures given by Hearn: according to a summary of 57 observations, 28 times the echinococcus existed in the lungs alone, 29 times it existed simultaneously in the liver, the spleen, the kidneys, the omentum, and the cæcum. Out of these 28 cases of isolated pulmonary localization, in 16 the hydatid was seated in the right lung, in 8 in the left lung, and in 6 both lungs were affected simultaneously.

Does the pulmonary tissue undergo any modification from the propinquity of the cystic pouch? This is a question of the greatest importance, whether from the point of view of prognosis or treatment. Here we find ourselves in presence of two contradictory affirmations. Walshe asserts that the intra-pulmonary cysts are scarcely ever seen in the midst of healthy tissue; according to him chronic pneumonia, or cirrhosis of the surrounding lung, is the rule. The Australian physicians, on the other hand, affirm that the cyst rarely determines serious alteration in the surrounding tissue. These contradictions may easily be explained. In Australia the disease is recognized from its very onset, and immediately treated by tapping; being thus arrested from the start in its evolution, it is quite natural that the echinococcus would not have the time to provoke a very severe irritation in the neighborhood of its point of implantation. This is not the case in Europe, where the affection is not diagnosticated till very tardily, and generally only after the expulsion of hydatid membranes, and where surgical intervention is not thought of till the last extremity. In these conditions the assertion of Walshe is absolutely true, and the most of the autopsies published in Europe show the lung invaded by the hydatid pouch, sometimes atrophied, flattened out, reduced to the condition of an envelope scarcely recognizable, superimposed on the adventi-

tious membrane, sometimes indurated, sclerosed, traversed by thick bands of fibrous tissue.

The development of the cyst may yet determine other lesions, more rare it is true, but also much more grave by reason of the frequently fatal complications which are the consequence; it is thus that are observed bronchial perforations, ruptures of the pleura, and ulcerations of some of the branches of the pulmonary artery.

Tuberculosis may also set in to complicate the evolution of the hydatid, and one can easily understand the difficulties which attend the diagnosis of such cases where the two diseases exist simultaneously.

CHAPTER XLIII.

CLINICAL DESCRIPTION.

For pulmonary cysts, as well as for cysts of the liver, we must evidently admit a latent period, or a period of tolerance during which the hydatid develops slowly without provoking any morbid reaction. The duration of this period is impossible to determine. There have been cases where the cyst was only recognized at the autopsy. Oftener, when the pulmonary hydatids were secondary, their development in the lung has been hardly noticed, the hydatid primarily localized in another organ, the liver or the kidneys, absorbing all the attention. On the other hand, even when localized primarily and solely in the lungs, the diagnosis may not be made (unless the cyst bursts into the bronchi), until at least it shall have attained a size sufficient to come in contact with the walls of the chest.

This period of progressive growth necessarily exists for every cyst, hence it will not do to admit, as do certain writers, two modes of invasion, one latent, and the other sudden.

The initial period is always latent; there arrives eventually a moment when the affection reveals itself by symptoms more or less striking, which forcibly attract the attention to the thorax.

We shall for convenience divide the evolution of the pulmonary cyst into three periods: a latent period; a stationary period (*period d'état*); and a period of complication, which ends in recovery or in death.

The first period, as the name indicates, contains no symptoms of any note or gravity. It may happen that the patient at this time has a little cough, or oppression; but nothing warrants referring these slight troubles to their true cause, even supposing that they engage the attention.

The symptoms of the second period may be divided into two groups: Symptoms of proximity, produced by the irritation of the pulmonary tissue in contact with the cyst, and special symptoms peculiar to the cyst itself.

Symptoms of Broncho-pulmonary Irritation.

These symptoms differ in nothing from those which are observed in every pulmonary affection; they may be referred to four principal heads: Cough, oppression, pain, hæmoptysis.

(*a*) **Cough.**—Writers have undertaken to attribute to the cough produced by pulmonary hydatids certain special characteristics; it has been said that it is convulsive, paroxysmal, coming on in fits, like those of whooping-cough. The truth is that the hydatid cough is as variable in its characters as the cough of any chronic pulmonary affection whatever. Habitually dry and short, like that of phthisis at the commencement, it may be soft, and accompanied with more or less expectoration of mucus or mucopus. In fine, in certain cases it may be particularly violent, fatiguing,

and take on a paroxysmal march; according to Bird, the cough characterized by fits belongs to the voluminous cysts occupying the bases of the lungs in the vicinity of the diaphragm.

(*b*) **Oppression and Pain.**—A dyspnœa more or less marked, and vague painful sensations in the chest, amounting even to "stitch in the side," are rarely wanting. It is apparent that the symptoms are in direct relation with the volume and with the rapidity of growth of the cyst. If this growth is very slow, the pulmonary tissue will accommodate itself easily to the parasitic tumor, and its presence will be tolerated so long as its dimensions do not too markedly impede respiration. If, on the contrary, the echinococcus rapidly takes on an exaggerated development, as is seen especially after traumatism, the dyspnœa and pain speedily acquire an alarming predominance.

Here, too, it is a fact that the large cysts of the base have the most direct and influential action in occasioning respiratory embarrassment and pain. In females, however, according to Bird, the oppression is much more marked when the cyst is seated in the upper parts of the lungs, a circumstance resulting evidently from the special respiratory type of the female.

In the cysts of the base of the right lung there have been noted as peculiar symptoms, pains radiating toward the shoulder, as in the case of cysts of the convex surface of the liver. But we shall soon see how difficult, not to say impossible, it is to decide if a cyst which occupies the base of the thorax on the right side is seated above or below the diaphragm. In general the hydatid at its stationary period determines only vague erratic painful sensations, without precise localization. As for the pungent pain of which Finsen speaks, it does not exist till some one of the complications of the third period supervenes.

(*c*) **Hæmoptyses.**—The spitting of blood is as frequent in hydatids of the lungs as in tuberculosis. At the period which we are considering, the mechanism of the hæmorrhages appears to be the same as in phthisis at the commencement. It is needful to remark, however, that abundant hæmoptyses are rare; Bird declares that he has seldom or never seen them. It is not then vomiting, but spitting of blood that one observes. The blood, of a lively red color, mingled with bronchial mucus, gives rise to an expectoration which resembles more the sputum of pulmonary cancer than that of simple pulmonary hæmorrhage. These expectorations of blood are repeated ordinarily at short intervals. In certain cases when the cyst has attained considerable dimensions, when tapping has been delayed, there may be a more free hæmoptysis; the blood is then raised almost pure, but the quantity does not exceed three or four ounces. Besides their diagnostic value, to which we shall return, these hæmorrhages have a certain importance in the evolution of the cyst. When they are too frequently repeated, and the quantity of blood rendered each time increases, there is reason to fear rupture of the hydatid pouch.

Physical Signs Indicating the Presence of the Cyst.

These signs are furnished by the inspection and palpation of the thorax, by percussion and auscultation. Inspection of the chest discloses in the majority of cases an enlargement of one of the sides of the thorax, or a bulging more or less clearly perceptible. When the cyst is seated in the inferior parts, the thoracic deformity is similar to that caused by large

pleuritic effusions, or voluminous cysts on the upper surface of the liver. The base of the thorax is dilated; the ribs are crowded outwards; the intercostal spaces widened, and as salient as the ribs themselves. When the hydatid occupies the superior lobes, it is under the clavicle that the bulging appears, generally at the external part. It is well to remember that the infra-clavicular bulging has less semeiological value on the left side than on the right; M. Woillez having demonstrated that in a fourth part of the subjects there exists on the anterior part of the left side of the thorax a normal projection which is exceptional on the right side.

On the level of the dilated thoracic region, palpation shows diminution or complete abolition of the vocal vibrations. Percussion gives an absolute dullness over the region occupied by the cysts, but the signs furnished by percussing in the neighborhood of the tumor have not been carefully studied. It is probable, however, that a tympanitic sound would be observed in certain parts of the thorax of the affected side; the presence of the cystic tumor putting the pulmonary tissue in the conditions necessary for the production of this percussion bruit.

Lastly, auscultation reveals complete absence of the respiratory sound over the dull region, while all around the vesicular murmur is normal, perhaps even a little puerile.

To recapitulate: localized bulging of the thorax with disappearance of vocal vibrations; complete dullness and absence of respiratory sounds, these phenomena being limited to a circumscribed point of the thorax; such are the physical signs of the pulmonary hydatid cyst in the stationary period. These signs are never plainer and more characteristic than when the hydatid is seated in one of the upper lobes, the dull and silent space occupying only a small extent, not larger than the palm of the hand, under the clavicle. It is readily intelligible that at the base, and especially in the posterior regions, the signs may be confounded with those of a pleuritic effusion.

As for Brainçon's sign, the hydatid thrill, which has in fact been but rarely noticed in hepatic cysts, it has never been observed in hydatids of the lungs.

But the most important particular, one which gives in some sort a pathognomonic value to the physical signs which we have just enumerated, and on which Dongan Bird has especially insisted, is the exact limitation of the dullness and respiratory silence. The dull space always presents a roundish contour, indicated by a line of demarcation so plain that one may trace it with ink or pencil; immediately beyond this line the sound becomes clear and normal, and no change in the position of the patient modifies it. Likewise the respiratory murmur, abolished in all the dull region, begins to be perceived as soon as you pass over the line marked by the ink—with a character slightly puerile, it is true—but indicating the integrity of the surrounding pulmonary tissue.

It is never the case that an encysted pleurisy, or any pulmonary inflammation or lesion whatever, offers so marked and abrupt a transition between the diseased parts and those which are sound. This characteristic belongs exclusively to hydatid cysts; and Bird gives it as a sign of almost absolute certitude.

But it is evident, in order that the value of this sign may be complete, that the cyst must not have determined in the pulmonary tissue any congestive or inflammatory process. Unfortunately if this freedom from phlogistic complications be the rule in Australia, where the attention of

physicians is constantly directed to the subject of this disease, these same conditions are rarely met in Europe, where hydatid cyst of the lungs is seldom diagnosticated till its third period, when complications of greater or less gravity appear, and singularly mask the signs proper to the tumor.

Period of Complications.

The complications which may result from the development of a hydatid tumor in the lungs are of three orders:

Congestive or inflammatory phenomena in the neighborhood of the cystic pouch; these may take place in the pulmonary tissue, in the pleura, or in the bronchi;

The inflammation and suppuration of the cyst;

The rupture of the sac into the interior of the bronchi or into the pleural cavity.

(*a*) **Pericystic Inflammatory Phenomena.**—An acute bronchitis with snoring and sibilant râles, sometimes unilateral, oftener double, is at times observed in subjects affected with pulmonary cyst, masking completely the signs which might indicate the existence of the cyst. What strikes the attention in these cases is the intensity of the dyspnœa, which is out of proportion to the signs of simple bronchitis. But it is the same here, as in cases of transient icterus supervening in the course of evolution of cysts of the liver before their rupture into the biliary passages; ordinarily the presence of the cyst is not recognized. The diagnosis of simple bronchitis is made. The symptoms, moreover, end in amelioration, and some more grave complication is needed to suggest the desirability of a more minute examination. This transient premonitory bronchitis must be distinguished from the purulent inflammation of the bronchi which follows the bursting of the pouch into the respiratory passages.

When the cyst in the progress of its growth impinges upon the pleura, it is apt to provoke the development of a pleurisy, sometimes dry, sometimes with effusion, whose signs conceal still more surely the existence of the hydatid tumor. As long as perforation does not take place, the pleurisy may remain fibrino-serous, and even get well. But what is oftener observed, is a congestive or inflammatory outbreak around the cyst, revealing itself to auscultation by foci of crepitant and sub-crepitant râles, foci which sometimes speedily clear up, but which at other times go on to hepatization, with tubular souffle and bronchophony; the sputa have a viscous sanguinolent character; paroxysms of fever ensue. We have observed a case of this kind where on three occasions the symptoms of broncho-pneumonia supervened, the disease extending itself by successive centres to the entire lung on the side where the hydatid was seated. Quain has reported a similar observation where the pneumonia existed on both sides, in a woman affected with cyst of the left lung. In this patient the pulmonary symptoms were accompanied by a very pronounced typhous state which gave reason to fear an attack of typhoid fever. The double pneumonia got well, but some months afterwards the cyst ruptured into the bronchi, and the patient coughed up numerous hydatid vesicles. In our patient, the cyst also ruptured some time after the inflammatory outbreak, but the rupture caused the almost sudden death of the patient. When, then, the phenomena of broncho-pneumonia are prolonged, or are repeated at short intervals, there is reason to apprehend the speedy rupture of the cyst.

(*b*) **Intracystic Suppuration.**—Suppurative inflammation of the hydatid sac ordinarily supervenes after the giving way of some part of its walls, and the characteristic expulsion of vesicles or membranes; the cystic cavity finds itself then in free communication with the air, and the pyogenic microbes contained in the bronchial secretions, and the penetration of these germs, easily explain the suppuration. There are cases where the cyst suppurates before rupturing and discharging, the patient voiding at the same time a flood of pus mingled with débris of hydatids. We must admit in explanation of these facts either that there are produced in some part of the cyst wall small fissures, insufficient to allow the contents of the vesicle to escape, but sufficient to give access to the inflammatory products which the bronchi contain, or that in undergoing multiplication in the adventitious membrane and the *membrana propria* of the cyst, the pus micrococcus has been able to penetrate to the interior. Certain occasional circumstances seem to favor this suppurative process, such as an acute attack of bronchitis or of acute pericystic broncho-pneumonia, a chill, a traumatism, especially when affecting the thoracic parietes.

An increase of the ordinary dyspnœa is then observed; this is accompanied by dull pains, at first deep seated, then more acute and lancinating on the diseased side. High fever sets in, which does not seem to present that character of intermittence which is so much remarked in suppurations of the liver; at least writers do not mention this feature. The suppurative process rapidly terminates by bursting of the cyst, which generally opens into the bronchi, being accompanied with all the symptoms habitual to purulent vomica, with this particular and pathognomonic character that the pus contains débris of vesicles or hydatid membranes.

(*c*) **Rupture of the Cyst and Cavity Formation.**—This is the ordinary termination of the evolution of hydatid cyst of the lungs. We have just seen that this rupture may be the consequence of suppuration of the cyst; but it may also be produced without previous inflammatory action by the exaggerated development of the cyst, by the progressive thinning out of its walls, the rupture ensuing after a strain, a fit of coughing, a blow on the chest, etc., sometimes without any known cause. In such cases the patient is suddenly taken with an extreme oppression, with a threatening suffocation, and after several violent paroxysms of coughing gives vent to a flood of liquid which fairly spurts from his mouth. This liquid is characteristic; it has a saltish taste very appreciable to the patient; it is clear, as limpid as spring water, and presents neither the viscosity nor the frothy appearance of the aqueous liquid ejected in certain cases of bronchorrhœa. Chemically it is not coagulable by heat, contains neither albumen nor phosphates, but a great proportion of chloride of sodium. The microscope, moreover, shows the hooklets isolated or in little masses, and the entire scolex of the parasite. There is not, however, generally need even of the microscope to detect the presence of the débris of the hydatid. It is rare in fact that the liquid does not contain echinococci, visible to the naked eye under the form of little white or grayish grains, like wheat, or little young vesicles whose size varies from that of a pea to that of a grape; there may be moreover whitish crispy shreds of the membrane peculiar to the hydatid. In other cases when the cyst has suppurated, the membranous débris and the vesicles are mingled with a purulent liquid and a limpid pus.

Finally, in a third class of cases, one or more attacks of hæmoptysis accompany the bursting of the sac and the expulsion of the hydatids,

whether this be from rupture of blood-vessels in the walls of the pouch, or whether some branch of the pulmonary artery or veins may have been involved in the bursting of the cyst.

With whatever variety of vomica you have to deal, the consecutive phenomena are the same. The quantity of liquid rendered is very variable; from a few ounces up to one or two quarts. Sometimes the vomica is formed at once; ordinarily, however, the cavity is filled and refilled at several different times, in a sort of intermittent and irregular manner. Often the patient continues for several weeks to expectorate on coughing little vesicles or shreds of membranes. Lastly, it is not impossible to find by the aid of the microscope, long after the formation of the first vomica, the characteristic hooks in the muco-purulent expectoration of the patient.

The consequences of the opening of the cyst into the bronchi are variable. It is, in the first place, the only mode of spontaneous cure of the pulmonary hydatid; the caseous and cretaceous transformations so common in the liver, do not seem to be observed in the lungs. In fact recovery quite often takes place as a consequence of the rupture. But death is not less frequent; it may come on quite suddenly at the very moment of the bursting of the cyst.

The irruption into the bronchi of a great quantity of liquid, and more likely still, the obstruction of a large bronchus or of the trachea by voluminous débris of the hydatid membranes, are without doubt the cause of this fatal termination. More generally the penetration of the bronchial liquids into the sac transforms the cyst into a vast abscess which never dries up, and whose persistent suppuration exhausts the patient, unless some septicæmic or gangrenous complication sets in to hasten the end.

It is useless to dwell on the physical signs observed in connection with the opening of the cyst into the bronchi. They are those of every pleuro-pneumonic cavity having communication with the outer air, that is to say,—all the signs of a large excavation; cavernous souffle, large bubbling, pectoriloquy; or when the cyst is very voluminous, those of an encysted hydro-pneumothorax; amphoric souffle, metallic tinkling, bruit of succession.

The bursting of the cyst into the pleural cavity is more rare than its rupture into the bronchi, which is due without doubt to the ready formation of adhesions at the point of contact with the sac. Perforation into the pleura may, however, take place, and this in two ways; sometimes the cyst bursts at the same time into the bronchi and into the pleura; sometimes the rupture is first into the pleura, then, at the end of a variable time, perforation into the bronchi ensues. The existence of communication solely between the cyst and the pleura, seems to us difficult of recognition; communication with the outer air not existing, one observes nothing but the signs of a pleural effusion. But these signs may belong to the cyst itself, when attended with pleuritic complication, or to a pleurisy developed by irritation in the vicinity, as well as to a pleurisy by perforation. On the other hand, even when a double pleural and bronchial fistula is produced, the signs of hydro-pneumothorax do not seem to us sufficient to assure the existence of perforation of the pleura, since these signs may be developed as a consequence of simple rupture into the bronchi when the cystic pouch presents a considerable development.

Nevertheless there is a certain quite peculiar phenomenon, which, according to Finsen, may enable one to affirm with great positiveness rupture of the cyst into the pleura. It is known that the bursting of a

hydatid cyst into the peritoneum, or the simple contact with that membrane of a few drops of the cystic liquid, as the result of a puncture, frequently determines the appearance of a general urticaria. It is the same with the pleura, according to the Iceland physician. In a doubtful case—a female patient presenting thoracic symptoms that were quite obscure, and who had never coughed up hydatids—Finsen on discovering one day a large eruption of urticaria on her body, did not hesitate to affirm the existence of a hydatid cyst communicating with the pleural cavity.

General Symptoms and March.

During the first two periods, hydatid of the lungs provokes no disturbance of the general health. Its development is absolutely silent, and apart from a little dyspnœa, a few attacks of bronchitis or pulmonary congestion, with or without hæmoptysis, nothing reveals the existence of the cyst unless it be a careful local examination. It is not so in the third period. Here the general phenomena assume an importance sufficiently great to sometimes lead to a mistaken diagnosis.

It may in a general way be said that the symptoms are those of pulmonary phthisis, or rather of chronic disease of the respiratory apparatus with suppuration. Whether in fact you have to deal with phthisis produced by the tubercle bacillus, or consumption determined by sclerosis of the lungs with dilatation of the bronchi, or by purulent pleurisy, by inhalation of irritant dust (pneumo-koniosis), the general aspect of the patient is the same. There is excessive emaciation and prostration, an irregular fever with evening exacerbations, night sweats and sometimes diarrhœa. A quite peculiar sign which is also observed in most chronic pulmonary diseases, and not in pulmonary tuberculosis alone, as is generally believed, is also met with in hydatid cysts of the lungs; I refer to the club-shaped Hippocratic fingers, where the nail curves inward on the palmar aspect of the finger, while the ungual phalanx takes on the characteristic drum-stick enlargement. This fact has been noted by Trousseau as well as by the Australian physicians.

An individual affected with pulmonary hydatid bears, then, a marked resemblance to a phthisical patient, and although bacillary phthisis often sets in to complicate the hydatid phthisis, it is well to bear in mind that the hydatid itself may, without the intervention of tuberculosis, determine the same general aspect; there may, however, be certain slight differences. Thus the diarrhœa and digestive troubles are generally wanting in hydatid phthisis; the patients keep ordinarily their appetite, and if fever sets in, it is not till the moment of the suppuration of the cyst, or after the opening of the latter into the bronchi or into the pleura.

The exaggerated growth which takes place in the cyst in certain cases, by the sole fact of the multiplication of hydatids, always provokes a general loss of strength, a marked debilitation, a certain degree of emaciation; the parasite absorbing to the profit of its development the products of nutrition destined for the regular performance of the functions of the economy. But the symptoms of the febrile cachexia, properly so called, are wanting, and there is at times between this so marked decline of the organism, without febrile troubles or alterations of the digestive functions, and the gravity of the pulmonary lesions, a contrast which ought to be utilized in the diagnosis between hydatids and tuberculous phthisis.

But when the walls of the cyst rupture, and there is a communication

between the sac and the outer air, the aggravation of the general symptoms becomes extreme.

The prostration is complete, accompanied by a hectic fever, which differs in nothing from that of tuberculous phthisis; the cough is incessant, the dyspnœa very marked; the patients continually expectorate copious sputa which are purulent, mixed with blood, and which contain débris of hydatids, half putrid and sometimes quite fetid. In these conditions observers have noticed partial gangrene of the lungs to set in. These symptoms of putrid suppuration and of destruction of the pulmonary tissue may be prolonged for several months, till the patient succumbs in a state of marasmus.

The fatal termination may be hastened by the giving way of a branch of the pulmonary artery. Thus it is that Waldeyer has recounted an observation where the patient after having spat up numerous hydatids, succumbed to abundant hæmorrhages. At the autopsy a large cystic cavity was found in the inferior lobe of the right lung, and rupture of a small branch of a pulmonary artery. Haberson has observed a similar case; it was a voluminous branch of the pulmonary veins which opened into the sac. These final hæmoptyses ought to be assimilated to hæmoptyses of the same kind which take place in the course of pulmonary tuberculosis, by rupture of one of those aneurisms of the pulmonary artery described by Rasmussen.

Diagnosis.

From reading the Australian authors, one would derive the impression that the diagnosis of hydatid cyst of the lungs is quite easy during the second period. At Melbourne or at Victoria, an individual who coughs and who spits blood while keeping the appearance of good health, is at once suspected of being affected with pulmonary hydatids. If the local examination shows a bulging with dullness and respiratory silence, the whole being really limited to a circumscribed region of the thorax, generally under the clavicle, there need be no hesitancy. But things do not present themselves thus in Europe, where we do not believe that any one has ever had occasion to diagnosticate the cyst before the period of complications. In these conditions the pulmonary hydatid may be confounded, 1, with tuberculosis of the lungs; 2, with encysted pleurisy; 3, with abscess of the lung; 4, with a tumor of the mediastinum; 5, with pulmonary cancer; 6, with hydatid of the liver. When the cyst is seated at the base of the right lung, the difficulty consists in determining whether the seat of the tumor is above or below the diaphragm.

Pulmonary Tuberculosis.—We have said that the general aspect of patients affected with pulmonary hydatids is often absolutely the same as that of persons suffering from tuberculous phthisis. The same cough with purulent expectoration, the same hæmoptysis, the same debility, with hectic fever, night sweats, etc., all combine to render the diagnosis perplexed and difficult. The slight differences which we have indicated, such as conservation of the appetite, the absence of diarrhœa, the less pronounced emaciation, besides the fact that they have nothing constant about them, are not sufficiently striking to substantiate a diagnosis. The local examination furnishes a better clue. The pulmonary hydatid can only simulate phthisis with excavation, or phthisis complicated with pneumothorax. Now in the first case, in tuberculosis at the period of excava-

tion, the thorax is generally retracted, and sunken in, the infra-clavicular hollows are depressed. The cyst, on the contrary, produces a dilatation of the chest, sometimes a clearly circumscribed bulging, which is never met with in a phthisical patient. On the other hand, in tuberculous hydro-pneumothorax, above the dull zone exists a zone of exaggerated sonorousness,—a tympanitic zone; the hydatid cyst, even after the penetration of air into its cavity, always gives a sound which is absolutely dull throughout its whole extent. But the pathognomonic sign is furnished by the study of the expectoration. If the patient raises vesicles, if by the aid of the microscope you detect hooks in the sputa, the diagnosis will present no difficulty. But it may be that you will be called upon to observe the patient at a moment when the sac is completely empty of its parasitic products. In these cases it is the presence or absence of Koch's bacillus with its characteristic reaction to certain staining liquids which will settle the diagnosis. It will not do, however, to forget that tuberculous phthisis may set in to complicate the hydatid phthisis, and that the existence of the bacillus does not absolutely prove the absence of the hydatid. It is plain that in cases as complex as these it must be left to the sagacity of the physician to establish the reciprocal value of all the signs observed.

Encysted Pleurisy.—Between an inter-lobar encysted pleurisy and a hydatid cyst, occupying the median lobe of the lung, there exists no difference as far as the physical signs are concerned. Percussion gives the same circumscribed dullness surrounded by parts having the normal resonance. The stethoscopic signs are the same before as after the opening of the sac. The two affections having an onset and a march equally insidious and almost latent, one cannot count much on any information supplied by the patient. If the cyst has not suppurated, the diagnosis will be apparent at the moment of the formation of a vomica with the expulsion of a clear and limpid liquid, or of vesicles which could only have belonged to a hydatid. But if the intra-cystic suppuration already exists, if the physician was not present at the time of the emptying of the purulent vomica, the confusion is likely to be inevitable. The vesicles, in fact, may be few, and infrequent, and of small dimensions; the membranes peculiar to the cyst may have been in great part destroyed by the suppurative inflammation, and only expelled in minute fragments which do not attract the attention of the patient. It will be necessary then to make a minute and repeated examination of the expectorated matters before pronouncing an opinion. In certain cases when the vomica already dates back several months, when the patient can furnish no precise information respecting the characters of this vomica, the diagnosis cannot go beyond that of encysted suppuration, there being nothing to warrant the affirmation of previous existence of hydatids.

Abscess of the Lung.—Abscess of the lung is an affection so rare that the thought of its possibility hardly need embarrass the clinician. Nevertheless when the rupture of the cyst has been preceded by those symptoms of acute pneumonia of which we have spoken, one may find himself perplexed. Here, however, the characters of the expectoration, and the presence of vesicles, of the débris of membranes, or of hooks, will alone permit an exact diagnosis.

Tumor of the Mediastinum.—When the cyst is seated in the anterior and superior parts of the thorax, the detection of a circumscribed dullness on the level of the sternum, or below the clavicle, coinciding with some of the signs of impediment to the venous circulation, may suggest a

tumor of the mediastinum. The most common tumors of this region are aneurism of the aorta, the glandular enlargements of adenitis or leucæmia, and malignant tumors. Each of these affections has special symptoms which do not belong to the hydatid cyst; it is, then, by exclusion that you arrive at a diagnosis of the hydatid, which is moreover the rarest of tumors observed in this locality.

Cancer of the Lung.—It would be difficult long to mistake hydatid tumor for cancer of the lung. There are certain signs, however, which are common to both these diseases, such as sanguinolent expectoration, repeated at short intervals, dullness limited to one part of the thorax, a state of general cachexia and enfeeblement. But the aspect of an individual affected with hydatid cyst has but a superficial resemblance to the cancerous cachexia. The march of cancer is, moreover, much more rapid than that of the hydatid. Lastly, it is rare in cancer of the lungs not to note glandular engorgements quite characteristic in the supra-clavicular hollow.

Cyst of the Convex Surface of the Liver.—When the cyst occupies the base of the right lung, it is almost impossible to decide whether you have to deal with a pulmonary cyst or a hydatid cyst of the liver projecting into the pleural cavity. This is known to be the habitual tendency of large cysts on the convex aspect of the liver; crowding back the diaphragm they sometimes rise to a considerable height in the right side of the chest. As a differential sign, authorities have given the curve with convexity turned upwards, which limits superiorly the line of dullness of the hepatic cyst. But if this sign has a certain value in the diagnosis of those hepatic cysts that are accompanied with effusion into the pleura, it has no value in the case with which we are now concerned, as the line of dullness of the pulmonary cyst presents the same disposition. When the cyst bursts into the bronchi, the diagnosis is sometimes facilitated by the character of the expectoration. If, in fact, the liquid comes from a hepatic cyst communicating through the diaphragm with a bronchial tube, it has a yellowish color, which becomes green by the addition of nitric acid, and which is due to admixture with the coloring matter of the bile; the microscope in certain cases will also show hepatic cells.

Prognosis and Treatment.

Hydatid cyst of the lungs is a grave affection, but this gravity seems to be much greater in Europe then in Australia. Out of 40 cases collected by Davaine, there were but 15 recoveries, 25 proving fatal. The statistics of Hearn are more favorable; out of his 144 observations, 60 ended in recovery, and 82 in death; but many of Hearn's cases were taken from the Australian journals. McGillivray and Dongan Bird do not assign to the disease quite so gloomy a prognosis. In their view there is more likely to be danger from the formation of new hydatid pouches in the lungs, or in some other organ, certain individuals possessing a certain aptitude for the fixation and germination of the parasite. But if the cyst is promptly diagnosticated, puncture with a fine trocar is almost sure to cause the death of the hydatid, and as far, at least, as puncture of the cyst is concerned, all fear of harm from the operation may be excluded.

Evidently it is to the delay in active surgical intervention that we must ascribe the greater gravity of hydatid cysts in our country. The hydatid is, in fact, generally left to itself, whether by reason of an erroneous diagnosis,

or through fear of interference. Now when left thus to its natural evolution, the pulmonary hydatid can only get well by finding an opening externally. This mode of cure is moreover quite frequent, since out of the sixty cases reported by Hearn which did not terminate in death, recovery took place by this mechanism in fifty-two. But it is none the less true that most of the disastrous complications, such as sudden death, persistent suppuration, pneumothorax, ulceration of an arterial branch, are also the consequence of this rupture of the cyst. It is, then, apparent that the object of the physician ought not to be to facilitate, or even patiently to wait so hazardous a termination, but rather to endeavor to avoid it by killing the parasite in the first stages of its development.

May this result be attained by means of medicines administered internally? We must emphatically answer no. Neither mercury, notwithstanding its indisputable parasiticide properties, nor turpentine, which has been recommended by reason of its well-known anthelmintic action, nor chloride of sodium, prescribed by Laennec, nor iodide of potassium, vaunted by Hawkins, have succeeded in curing hydatid cyst of the lungs. Bird has no faith in the efficacy of medical means till after puncture of the cyst, when the hydatid having survived the incomplete evacuation of its liquid contents, yet finds itself considerably enfeebled in its vitality. Then only he concedes a certain value to a limited number of medicinal agents, and in particular to turpentine and bromide of potassium.

Surgical Treatment.

Hence the only curative treatment of the hydatid is early puncture of the cyst. As soon as they notice a bulging, with dullness clearly circumscribed, in an individual who has had several slight attacks of hæmoptysis, the Australian physicians diagnosticate a hydatid cyst, and make the puncture. Bird employs a simple trochar of the smallest diameter; he regards aspiration as useless, the perfectly limpid liquid of the young cyst flowing freely of itself, without the necessity of facilitating this flow by the aid of the aspirator vacuum. He, however, recognizes the fact that the parasite is the more certain to die the more completely the liquid is evacuated, and in fact, as the modern aspirator prevents the germs of the air from entering the cyst, we are inclined to think that puncture with the large needle of Dieulafoy's or Potain's apparatus ought to be preferred to puncture by the simple trochar. We should, however, make reservation in the case of cysts of large dimensions. When the sac has attained a considerable size, its walls come into close relation with bronchi often of fine calibre, which are slowly destroyed by the evolution of the cyst. As long as the sac is full of liquid there is equilibrium between the interior pressure of the cyst and the atmospheric pressure exercised by the bronchial tubes. But if this equilibrium be too suddenly broken, if, in other words, the pressure within the cyst be too much lessened by the aspirator, there is reason to fear rupture of the bronchial walls in places where pressure from without is principally exerted, and there is danger of producing the very vomica which it was sought to avoid by the puncture. M. Gibier, who proposed this ingenious explanation, has in fact witnessed, in a case communicated to the Anatomical Society, the rupture of the cyst during a puncture made with the aspirator needle, and the patient succumbed, suffocated by the irruption of liquid into the bronchi. The procedure of Dongan Bird ought, then, to be adopted in the cases of

voluminous cysts, and puncture with the simple trochar preferred to puncture with the aspirator.

The electrical treatment by constant currents, by the aid of needles introduced into the cyst, has thus far only been applied to hydatids of the liver, and the results are not yet sufficiently assuring to warrant its application to hydatid of the lungs.

At a more advanced period when the cyst suppurates, whether communicating with the bronchi or with the pleura, and when there is no longer anything but a focus of putrid decomposition, punctures become absolutely useless. It is necessary then to introduce a large trochar and leave a permanent drainage-tube by which the putrid liquids shall find issue. This drainage-tube will serve at the same time for disinfectant injections. It may, however, be better to act as in old cases of purulent pleurisy, and perform the operation for empyema, to give a large vent to the débris of the vesicles and putrefying membranes which keep up suppuration in the sac.

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APPENDICES.

APPENDIX A.

THE

PNEUMONIA-COCCUS OF FRIEDLÄNDER.

(*Micrococcus Pasteuri*, Sternberg.)[1]

BY

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SURGEON U. S. ARMY.

In this paper I desire to call attention to the so-called pneumonia-coccus of Friedländer, which I shall take the liberty of naming *Micrococcus Pasteuri*. My right to name the micrococcus discovered by Friedländer in the exudate of croupous pneumonia must depend upon my ability to make good the claim which I here state, viz.: that the pneumonia-coccus of this author is, in fact, identical specifically with a micrococcus previously described by me, which is found in normal human saliva, and with that found by Pasteur in the blood of rabbits which had been injected with the saliva of a child which died of hydrophobia in one of the Paris hospitals.[2]

In attaching to this micrococcus the name of the illustrious French chemist I have no desire to perpetuate the memory of the mistake he made in supposing for a time that it was the germ of hydrophobia. Having found that this was a mistake he did not fail to correct it, so no doubt Koch will do, if he has made a mistake in announcing his "comma bacillus" as the much sought cholera germ. It is easy to make mistakes in this field of investigation; easier, perhaps, than to acknowledge them. And believing, as I do, in human fallibility, I have no hesitation in questioning the conclusions of the most illustrious workers in the field of microbiology, if they are in conflict with my own observations. On the other hand, if upon fuller investigation I am convinced that I have been mistaken in regard to this or any other question, I shall feel no hesitation in following the example of Pasteur in making a public acknowledgment of my error. At all events the name will stand for the oval micrococcus which produces a fatal form of septicæmia in rabbits, and which is constantly present in my buccal secretions.

If I am right as to the specific identity of this micrococcus with the

[1] Read before the Pathological Society of Philadelphia, April 17, 1885. Published in the American Journal of the Medical Sciences, Philadelphia, July, 1885.

[2] Comptes rendus Ac. de Sc., 1881, xcii. p. 159.

micrococcus discovered by Friedländer in the exudate of croupous pneumonia, it does not follow that Friedländer is wrong in assigning to this organism an etiological rôle in connection with this disease. This is a question which I will not discuss at the present time, as I hope to make it the subject of experimental inquiry at as early a date as practicable.

In giving a specific name to a micrococcus which I first observed nearly five years ago, and which I have repeatedly studied by means of oil immersion objectives—the one-eighteenth and one-twelfth inch hom. im. of Zeiss—by the most approved methods of staining and cultivation, and by numerous experiments upon animals, I can scarcely be accused of undue haste. And inasmuch as this is the first micro-organism which I have attempted to name among the multitude which I have encountered in the course of my bacteriological studies, I may be acquitted of any special proclivity for conferring names upon supposed "new species," as is the fashion among amateur naturalists.

I have heretofore spoken of this particular micrococcus as "the micrococcus of septicæmia in the rabbit;" but as there is more than one form of infectious septicæmia in the rabbit known to us by laboratory experiments, each due to a different micro-organism, this term is evidently inexact. Moreover, it would indicate that this particular micrococcus finds its usual habitat in the blood of rabbits affected with the form of septicæmia to which it gives rise. This is by no means true, for the organism in question is widely distributed, and it is only by inoculation experiments that the fact has been developed that it is a pathogenic species, so far as rabbits are concerned. I made this discovery in the summer of 1880, quite accidentally, having injected a little of my own saliva under the skin of a rabbit, as a comparative experiment, to ascertain whether a fluid supposed to be innocuous would give rise to any febrile disturbance. The promptly fatal result, and the presence of a multitude of oval organisms in the blood, at once aroused my attention, and as is well known I have since made numerous additional experiments, at different times and places, and always[1] with the same result: The animals die in from 24 to 48 hours; they present the same pathological appearances; their blood is infectious in the smallest quantity, and it contains vast numbers of the oval micrococcus which I now name *Micrococcus Pasteuri*.

Upon making sections of the tissues of a rabbit dead from this form of septicæmia, the oval micrococci are found in great numbers in the capillaries of the various organs, as I am prepared to show your microscopical experts in slides which I have brought with me. Repeatedly I have produced this infectious disease in rabbits by injecting beneath their skin pure cultures of the micrococcus in question.

All this is a matter of record, and the experimental proof is as definite as is that offered by Koch in regard to the fatal form of septicæmia in mice which he has so well studied, or in the better known infectious disease, anthrax. Yet in spite of my detailed record of experiments made, of my frequent repetition of these experiments, and of my photographs from nature which illustrate my first paper, published in 1881, and the article upon septicæmia in rabbits in my book,[2] which was published more than a year ago, I find in Klein's recent work[3] the following reference to my experiments:—

[1] In a few instances only, out of a large number of experiments, have rabbits survived the injection subcutaneously of a few minims of my saliva.

[2] Bacteria. Wm. Wood & Co., New York, 1884.

[3] Micro-organisms and Disease. 1885.

"That saliva of the healthy dog and of man inoculated subcutaneously into rabbits sometimes produces death in these animals [Senator] had entirely escaped his [Pasteur's] notice. Sternberg has proved this in an extensive series of experiments. His own saliva proved sometimes fatal to rabbits. They die of what is called septicæmia, and Sternberg thinks it due to the micrococci; but this is not to be considered as proved."

I claim that the fact is proved, and that the experimental evidence of this has been upon record for nearly four years, and I am prepared to repeat the experiments, and to demonstrate that such is the case. I look upon the fact that an infectious disease of a lower animal may be induced by inoculation with a micro-organism which is habitually found as a

FIG. 1.

FIG. 2.

FIG. 3.

FIG. 4.

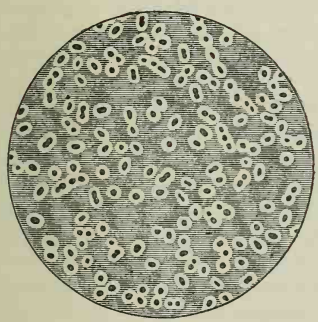

FIG. 5.

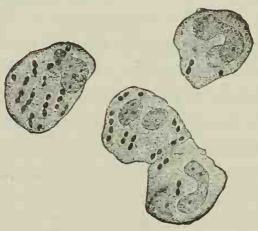

FIG. 6.

FIG. 1.—Micrococcus Pasteuri from blood of rabbit inoculated subcutaneously with normal human saliva (Dr. S.). Stained by the method recommended by Friedländer. Magnified 1000 diameters.

FIG. 2.—Micrococcus Pasteuri from blood of rabbit inoculated subcutaneously with fresh pneumonic sputum from a patient in the seventh day of the disease. Same staining and amplifications as Fig. 1.

FIG. 3.—Surface culture of M. Pasteuri showing development of long chains. Same staining and amplification.

FIG. 4.—Surface culture of M. Pasteuri from blood of rabbit injected with pneumonic sputum, showing the so-called "capsule" of Friedländer. Same staining and amplification.

FIG. 5.—From a photo-micrograph made by the author of this paper in 1881, and used in April of that year to illustrate a paper on "A fatal form of septicæmia in the rabbit induced by the subcutaneous injection of human saliva." The preparation is from the blood of a rabbit recently dead, and is stained with an aqueous solution of iodine and potassic iodide. Magnified 1000 diameters. Photographed with Zeiss's one eighteenth inch hom. ol. im. objective.

FIG. 6.—Copied from illustration accompanying the paper of Salvioli in the Archivio per le Scienze Mediche, Turin, vol. viii., Tar. viii. Fig. 2. "Cells of the pleuritic exudation containing pneumonia cocci, mounted in Canada balsam." Stained with gentian violet. Amplification not stated (about 1000 G. M. S.).

harmless parasite in the mouth of man, as something more than a curious circumstance to be recorded and forgotten, and as having an important bearing upon vital questions relating to the genesis of "disease germs," questions which are now largely occupying the attention of leading pathologists in all parts of the world. But in the discussion of these questions I must insist that experimental evidence obtained on this side of the Atlantic is entitled to just as much consideration as that which comes to

us across the water. And unless the truth of my detailed account of experiments made is impeached, I can only account for such a verdict as that which Klein has recorded in the sentence above quoted, upon the supposition that he has not read the evidence presented, or that he is controlled by a geographical bias, unworthy a true student of science, in estimating its value.

In the blood of rabbits recently dead, and in fresh cultures, *Micrococcus Pasteuri* presents the appearance shown in Figs. 1, 2, and 5. The oval elements vary considerably in their dimensions, and especially in length, and are seen solitary, in pairs, or in chains of three or four. When undergoing rapid multiplication, they are commonly in pairs, but in surface cultures long chains are often formed, as seen in Fig. 3. In these the individual cocci approach more nearly to a spherical form, than in the pairs encountered in the blood of a rabbit just dead, where they are often so much elongated as to appear rod-shaped. My observations indicate that these rod-like forms are upon the point of division into two pairs of oval elements, and, indeed, upon close inspection with high powers, the commencement of transverse division may be recognized in each element of the pair. Morphologically these pairs of oval or rod-like elements closely resemble the ordinary bacteria of putrefaction —*B. termo*, and might easily be mistaken for this organism. The supposition that they are identical is easily disposed of. Our micrococcus is, under all circumstances, motionless, while *Bacterium termo* is an actively motile organism. *Micrococcus Pasteuri* is difficult to cultivate, being exceedingly sensitive as regards the composition and reaction of the culture medium. It does not give rise to any putrefactive odor in fluids in which it is present in the greatest abundance. In liquid cultures it only causes a slight opalescence of the culture medium, and its presence upon the surface of Koch's "flesh-peptone-gelatine" can only be detected by close observation. It does not liquefy the gelatine, and does not grow readily on the agar-agar gelatine. In blood serum and in liquid culture media, it is seen with difficulty under the microscope, owing to the fact that its refractive index does not differ greatly from that of the fluid containing it. On the other hand *B. termo* thrives in any medium containing gelatine or albumen, and causes this to undergo putrefactive decomposition.

The pneumonia coccus of Friedländer is also described as oval in form, or as being found in pairs and in short chains. According to Talamon, "it has the form of a grain of wheat, of from 1 to 4 μ. in length, and one-half to 1 μ. in diameter. It is isolated, more frequently in pairs, and sometimes in chains of four. This corresponds with the description I have given of *M. Pasteuri*. During the past winter I have made the acquaintance of this micrococcus as it appears in pneumonic sputum, and I find that morphologically it does not differ from my *Micrococcus Pasteuri*.

But how about the "capsule" which Friedländer has described as the distinguishing character of pneumonia-coccus? Dr. Friedländer's first communication was made to the Medical Society of Berlin, on the 19th November, 1883, and this capsule was described as something quite new, and as characteristic of the pneumonia-coccus. I shall show you presently upon the screen a photo-micrograph (see Fig. 6), made by myself in the spring of 1881, in which oval micrococci in pairs and chains of three or four elements are surrounded by a capsule exactly resembling that described by Friedländer in his communication made two years later. This photo-

micrograph was reproduced by the heliotype process, and appears as one of the illustrations which accompany my paper "on a fatal form of septicæmia in the rabbit," published in vol. ii. no. 2, of *Studies from the Bibliographical Laboratory* of Johns Hopkins University. This journal, however, is not likely to come under the eye of medical men, and it is not surprising that the paper referred to had escaped the attention of Friedländer. The same paper was published in the *Bulletin of the National Board of Health*, of April 30, 1881, but without the heliotype illustration. The micrococci with "capsules," are also shown in Fig. 3, of Plate VI., in my book on "Bacteria," published in 1884.

Unfortunately a mistake has been made in the descriptive text facing this plate in my book, where the statement is made that the micrococci in this figure were stained with methyl-violet. This mistake was due to a fault in my memory, and to a failure to refer to my original paper published more than two years before, where the statement is clearly made that "in these photographs the staining was effected with iodine solution" (p. 196).

There can be no question as to the morphological identity of the micrococcus represented in this photo-micrograph with that described by Friedländer, and the capsule was brought out in the specimen from which this photo-micrograph was made by the use of the same staining solution which Friedländer recommends for its demonstration, viz., a weak aqueous solution of iodine dissolved by the aid of potassic iodide. In my paper referred to I say, "In these photographs the staining was effected with iodine solution," and a little further on I give the formula as follows, "(iodine, grains iii, potassic iodide, grains v, distilled water, grains 200)." In the same paper I give the following account of this envelope of mucus material which Friedländer describes as a capsule.

"The most striking morphological difference between the micrococcus as shown in Figures 2, 3, and 4, and in Figure 1, is the aureole which surrounds the well-defined dark central portion in the latter figure.

"Pasteur says of this appearance: 'This organism is sometimes so small that it may escape a superficial observation. . . . It is an extremely short rod, a little compressed towards the centre, resembling a figure 8. . . . *Each of these little particles is surrounded at a certain focus with a sort of aureole which corresponds perhaps to a material substance.*"'

Pasteur's inference that this aureole represents a material substance, and is not simply the result of diffraction, is fully sustained by my observations and my photographs. The slighter aureole seen in Figures 2 and 3 is probably a result of diffraction; but the use of aniline violet as a staining fluid promptly demonstrates that in Figure 1 we have to do with a material substance. The refractive index of this substance must be very nearly that of blood serum, for it is with great difficulty that this aureole can be distinguished without the aid of staining material. It may be seen by the practised eye with a good immersion lens, but, as already mentioned, even the darker central portion, which alone is seen at first, may easily escape observation, and a false impression is obtained as regards the real size of the organism. When, however, a small drop of blood, dried upon a thin glass cover, is immersed for a minute or two in a solution of aniline violet, and then washed and examined with, even, a good one-fifth-inch objective, the observer will be astonished to find a multitude of organisms, solitary, in pairs, and in chains, having a diameter of more than 1 μ. and mostly possessing an oval or elongated form, which might

lead to the inference that they should be referred to the genus *Bacterium*, Duj., rather than to *Micrococcus*, Cohn.

"The reason of this apparent change in dimensions as the result of staining, is that the substance which constitutes the almost invisible aureole is deeply stained by the aniline, and the central portion, which was before seen because of its highly refractive index, is now lost to view in the uniform and deep violet color which the whole organism possesses."

Friedländer says with regard to his so-called capsule, that several microchemical tests justify the conclusion that it consists of mucine, or of a substance resembling mucine. His account shows that it is not constantly present, and varies greatly as to the extent of its development. In this his pneumonia-coccus also corresponds with my *Micrococcus Pasteuri*. The development of this external envelope of mucine to the extent shown in my photo-micrograph is altogether exceptional. I have not yet ascertained the exact conditions which control the development of this envelope, but believe it to be most marked in a rich culture-medium, and as a result of an exceptionally vigorous and rapid development of the micrococcus. Friedländer says: "The formation of capsules appears to be an attribute of the pneumonia cocci at the highest point of their development."[1]

Prof. Germain Sée, in his recent work,[2] after giving an account of Friedländer's method of staining, says:—

"Friedländer attaches such an importance to the capsule of the micrococcus that he declares that it is never wanting in acute fibrinous pneumonia, and that in all the other forms of pneumonia studied by him he has only found cocci without capsules. Nevertheless we must hasten to say, this capsule has no value whatever. Talamon does not refer to it. According to him it does not belong to the coccus; it is the result of the mode of preparation and staining of the exudate. It is met with in various microbes found in bronchial mucus treated by the method of Friedländer. This is also the opinion of Professor Cornil. He says: "We believe, M. Afanassiew and myself, that the appearance is due simply to a refraction of the liquid around the micro-organism, and that the capsules are produced artificially.' On the other hand, Fraenkel, at the Medical Congress in Berlin of 1884, declares that the formation of the capsule is not a constant phenomenon; that this capsule is formed in other schizomycetes; finally that it is not possible to consider the capsules as characteristic of the micrococcus of pneumonia. Friedländer himself, at the same Congress, replying to Fraenkel, has given the last blow to this famous capsule, in admitting that which characterizes the micrococcus of pneumonia is neither the capsules, nor its cultures in nail-form, but the sum of the morbid phenomena which it determines. There should then be no further question of micrococci with capsules. It is the form of the microphyte which ought to be taken as characteristic, joined to its pathologic effects as shown by inoculation."

A second distinguishing character assigned by Friedländer to his pneumonia-coccus was its peculiar nail-like mode of growth in gelatine cultures. Klein has made the curious mistake of speaking of the coccus itself as nail-shaped. In his recent work,[3] he says (p. 329), "They are

[1] From a review by Warwick in Centralbl. f. cl. med. Wissench., Berl. 1884, No. 7, p. 107.

[2] Vide ante, p. 58.

[3] Micro-organisms and Disease, London, 1885.

oval, of *a peculiar nail-like shape*, about 0.001 mm. long," etc.; and again on the following page: "The micrococci were of *the peculiar nail-like shape*, and were characterized by a mucinous capsule."

Prof. Sée, in the work above referred to, shows that the nail-like growth in gelatine cultures is after all not a character of any great value, and that the body of the nail is formed by the growth of the organism along the track of the inoculating needle, and the head by a little knob upon the surface of the gelatine at the point of entrance. Professor Sée says:—

"According to Friedländer the cultures have a peculiar aspect: upon the surface of the gelatine a little elevation is formed from which a whitish substance extends downwards into the nutrient gelatine. This is what the German author speaks of as a nail-shaped culture. The coccus develops at the temperature of the chamber and does not liquefy the gelatine. Here again Friedländer has been mistaken, in describing this aspect of the culture as a peculiarity of the pneumonia microbe. The nail form does not depend upon the organism but upon the manner in which one introduces it into the gelatine-peptone, by means of a platinum wire." (Vide ante, p. 53.)

At the same time that Friedländer was pursuing his researches with reference to the etiology of pneumonia in Berlin, Talamon was engaged in similar studies in the laboratory of the Hôtel-Dieu in Paris. His first communication was made to the Anatomical Society, November 30, 1883, a few days after Friedländer's communication to the Medical Society of Berlin. This author has nothing to say about a capsule. The morphology of the organism found by him in the pneumonic exudation, drawn from the lung during life by means of a hypodermic syringe, or obtained post-mortem, corresponds with that of my *Micrococcus Pasteuri*. Talamon experimented upon twenty rabbits, two guinea-pigs, and four dogs. Injections of liquid cultures directly into the pulmonary tissue of the guinea-pigs and dogs gave a negative result. Of twenty rabbits inoculated in the same manner with the exudate from cases of pneumonia, or with liquid cultures, four recovered after showing symptoms of being sick for five or six days: the others died. Of those which succumbed, two presented no alterations of the lungs, which were scarcely congested, but a "fibrino-serous pleurisy" and fibrinous pericarditis was found. In eight rabbits there was fibrinous pneumonia, sometimes involving an entire lung, sometimes only the inferior lobe. In these cases also there was pleurisy and pericarditis with fibrinous and serous exudation. The blood of the rabbit contained the oval micrococcus in abundance.

I call attention here to the fact reported in my first paper on "a fatal form of septicæmia in the rabbit," viz.: that the guinea-pig is less susceptible than the rabbit, and that the dog shows no susceptibility. Nevertheless two out of three of my guinea-pigs succumbed, one in three and one in seven days, while Talamon's guinea-pigs did not die. Moreover, Talamon injected six rabbits subcutaneously, and these all withstood the injection and showed no special indications of ill-health. If then the organism injected was the same as that which I have found to kill rabbits almost without fail, within forty-eight hours, we must admit at least that is has not the same pathogenic potency. My own observations lead me to believe that this micrococcus when obtained from the same source differs in pathogenic powers at different times, and that while probably present

in the saliva of healthy persons generally, it has not in all cases the same degree of pathogenic power. Thus is my original experiments, I report as follows:—

"The saliva of four students, residents of Baltimore (in March), gave negative results; eleven rabbits injected with the saliva of six individuals in Philadelphia (in January) gave eight deaths and three negative results; but in the fatal cases, a less degree of virulence was shown in six cases by a more prolonged period between the date of injection and the date of death. This was three days in one, four days in four, and seven days in one."

I also mention the fact that the buccal secretions of two Cuban gentlemen, medical students in Philadelphia, proved to be quite as virulent when injected beneath the skin of rabbits as my own. This, taken in connection with the circumstance that I have spent much time in the South, led me to think that possibly this unusual degree of pathogenic power might be due to the fact that the variety of the micrococcus to which it is due, and which is constantly in cultivation in my mouth, came from a southern latitude. This supposition receives some support from the experiment of Claxton [1] made in this city, at the University of Pennsylvania, in 1882. The results reported were in general in conformity with those previously reported by myself, but they showed that the saliva of all persons does not exhibit the same pathogenic potency, as manifested by its effects upon rabbits, for of eighteen rabbits injected with the saliva of seven individuals, four recovered, five died in less than five days, four in five to ten days, and four in ten to fifteen days. Claxton remarks: "I also have observed the same remarkable virulence in the residents of tropical countries, as Cuba and Brazil, as also in the case of negroes, the saliva of which latter race exhibits it to an extreme degree."

The difference in pathogenic power shown by this oval micrococcus as found in the buccal secretions of healthy individuals, corresponds with a difference in the results obtained by three different experimenters who have injected pneumonic sputum into rabbits, in different latitudes. Thus Friedländer reports a negative result from nine injections made through the thoracic walls, while Talamon had sixteen fatal cases out of twenty injected, and Salvioli,[2] whose experiments were made in Italy, reports a uniformly fatal result, within forty-eight hours, from similar injections made into four rabbits. The greater pathogenic power of the material injected by Salvioli, is shown by the fact that his rabbits all died within forty-eight hours, and also by a uniformly fatal result from injections into guinea pigs (9). The pathological appearances resulting from the injections practised in these two species of animals were identical, and correspond with those which I had previously obtained from the injection of normal human saliva. All of the authors mentioned refer to tumefaction of the spleen, as a constant result. Both Talamon and Salvioli refer to the abundant presence of the oval micrococcus in the blood, and effused serum in the pleural cavity, of animals which have succumbed to the injections made. The paper of Salvioli is illustrated by some very well-drawn figures, which illustrate the morphology of the organism found by him in the pneumonic exudate and in the blood of animals into which

[1] Philadelphia Medical Times, June 17, 1882.

[2] G. Salvioli. Natura infettiva della pulmonite crupale. Arch. per le Scienze Mediche, vol. viii. pp. 127-148 (1884).

this has been injected. Fig. 7 is copied accurately from this author's Fig. 2, and shows that his pneumonia coccus is identical morphologically with my *Micrococcus Pasteuri*. The results obtained by the author last mentioned, like those of Talamon and of Friedländer, are supposed by their author to establish the fact that croupous pneumonia is an infectious disease. Without denying this fact, we submit that these results are in conformity with the claim made in the present paper, as to the specific identity of the oval micrococcus found constantly in the pneumonic exudate, with the micrococcus found in normal human saliva which produces an infectious form of septicæmia in rabbits. In support of my claim as to the physiological identity of the oval micrococci from these two sources I offer the following literal translation of the experiments of Salvioli upon rabbits:—

"*Jan.* 17, 3 *P.M.*—Rabbit weighing 1970 grammes; very thin when purchased. Injected into right thoracic cavity, 1.5 c.c. of the usual exudation. The rabbit died at 10 A.M. on the 19th. At the post-mortem examination made immediately after death we noted: collection of turbid serum in the right pleural cavity which coagulated after being drawn. Fibrinous pseudo-membrane upon the surface of the lung, especially the inferior lobe. Lung, especially the inferior lobe, enlarged and congested, contains little air. Upon section a little serum escapes from the surface. Spleen congested. Characteristic pneumonia cocci in the pleuritic exudation; not in spleen. Upon microscopical examination of an indurated piece of lung, micrococci were found in the costal and visceral pleuræ, and in the pulmonary tissue, presumably between the lymphatics; and in the pulmonary alveoli. Near the pleura a slight collection of fibrinous exudation."

"*Jan.* 20, 3 *P. M.*—Rabbit weighing 2080 grammes, of Sardinian race, of exceptional resistance, and therefore adapted for the experiment. Injected into right thoracic cavity 1 c.c. of the exudate from the dog"—killed at a previous experiment.

"At noon on the 21st the rabbit was found dead. At the autopsy a turbid liquid, rich in pneumonia cocci, was found in the right pleural cavity, infiltration of the mediastinal cellular tissue by a turbid serum rich in cocci, fibrinous pseudo-membrane upon the right pleura. Spleen enlarged, and with cocci in its pulp. At the examination of a hardened specimen, cocci were found in the pleura, visceral as well as parietal, and also in the pulmonary tissue, in the vicinity of the pleura. In this also was found a little of the fibrinous exudation in the alveoli."

"On the 28th I received another specimen of exudate from the medical clinic, which had been extracted [1] from a patient with fibrinous pleuropneumonia, in the twelfth day of sickness. The slightly turbid liquid had an alkaline reaction and pale yellow color. When allowed to stand a sediment was deposited corresponding to one-eighth of the liquid column. A microsocopic examination of the sediment showed the presence of leucocytes, red globules, a quantity of granular material, and some very rare pneumonia cocci, but slightly stained, notwithstanding a repetition of the preparation; instead I observed some smaller granules which did not take the color, which resisted drying, and which I have called spores. This exudate I injected into two guinea-pigs, two rabbits, and two dogs. *Only*

[1] I understand that the liquid was extracted from the pleural cavity by an aspirating syringe, although this is not distinctly stated.—G. M. S.

the rabbits gave me a favorable result.[1] The other animals survived and did not present the slightest disturbance."

"*Jan.* 28, 6 *P. M.*—Rabbit weighing 1920 grammes; injected into right thoracic cavity 1 c.c. of the above serum after mixing up the sediment. The rabbit was found dead the morning of the 29th, and at the autopsy was found an abundant, turbid, sero-fibrinous exudation in the right pleural cavity, fibrinous pseudo-membrane upon the pleura. The cellular tissue of the mediastinum was infiltrated with turbid serum; parenchyma of right lung partly deprived of air by compression; with this exception normal:" free translation for "*del resto nulla di particolare.*" "Spleen enlarged; pneumonia cocci in the pleuritic exudation and in the splenic pulp. At the microscopic examination of a hardened piece, the pneumonia cocci were found in the pleura, in the pulmonary tissue also, at a distance from the pleura.[2] In some alveoli in the vicinity of pleura, was found a fibrinous exudation.

Jan. 28, 2 *P. M.*—Rabbit weighing 1890 grammes; injected into right thoracic cavity 1 c.c. of the limpid serum above referred to. On the morning of the 31st the rabbit died, and at the autopsy was found: turbid exudation in right pleural cavity, fibrinous pseudo-membrane upon the pleura, right lung enlarged, resistant, non-crepitant; the inferior lobe presented the aspect of red hepatization, pronounced tumefaction of the spleen. The characteristic pneumonia cocci were found in the exudate, in the pulmonary juice, and in the splenic pulp."[3]

I shall now record some recent experiments made by myself.

January 2, 1885.—Inoculated two rabbits, subcutaneously, with pneumonic sputum, collected with great care by my friend, Dr. Rohé, and brought to me at once—from a white male patient, aged 19, in the seventh day of illness, second day of bloody expectoration. Both rabbits were found dead on the morning of January 4. In both the pathological appearances were identical with those constantly observed by me in rabbits killed by the subcutaneous injection of my own saliva, viz., extensive inflammatory œdema extending from point of injection, enlarged spleen, and presence of oval micrococci in blood and in effused serum in the subcutaneous connective tissue. Fig. 2 is drawn from a slide mounted at the time, and represents this micrococcus as seen in the blood.[4]

Remarks.—It may be asked whether possibly my *Micrococcus Pasteuri* may not have been present, accidentally, in the pneumonic sputum, as a result of the fact that the exudate into the alveoli of the lungs must come in contact with the buccal secretions of the patient when it is expectorated. In reply to this supposition I would say that I have here a slide, which I shall be happy to submit for the inspection of the microscopical experts present, which shows the capsule of Friedländer in a most beautiful and unmistakable manner. This slide is from a surface culture which was started from the blood of one of these rabbits inoculated with pneumonic sputum. (See Fig. 4.) I cannot, then, doubt that this is in truth the micrococcus described by Friedländer, and as in the blood of the rabbits, it was identical morphologically with my *Micrococcus Pasteuri* (com-

[1] Italics by translator.

[2] They had invaded the blood and were distributed through the capillaries generally—G. M. S.

[3] Op. cit., p. 133.

[4] This and the other figures illustrating the present paper are from camera lucida drawings made by my laboratory assistant, Dr. A. C. Abbott, of Baltimore.

pare Figs. 1 and 2) and shows identical physiological characters when injected beneath the skin of these animals, I feel justified in assuming the specific identity of the micrococci from the two sources.

March 14.—Four rabbits were injected subcutaneously with a small quantity (about 1 c.c.) of my own saliva. Two of these rabbits were found dead on the morning of March 16, and the other two died during the morning. All presented the usual pathological appearances so often described, viz., enlarged spleen, extensive subcutaneous œdema, slight hemorrhagic extravasations, etc. The blood of all of these rabbits contained the oval micrococcus in abundance. (Fig. 1 is drawn from a preparation mounted at the time.) Cultures were started from this blood, and served for the following experiment.

March 31.—I injected into the thoracic cavity of two rabbits, by means of a hypodermic syringe, a small quantity of a culture of *M. Pasteuri*, in Koch's flesh-peptone gelatine. Both rabbits were found dead the following morning. In both the pleural cavity contained turbid serum, and there was a fibrinous deposit upon the surface of the lung on the side (right) into which the injection was made. The margin of the lung in one appeared congested, but there was no inflammation of the lungs, which floated in water.

Remarks.—Death occurred in these cases more promptly than when I have injected the same material subcutaneously. This is not surprising, for it seems probable that the blood would be invaded by the parasite more promptly when it is thrown into the pleural cavity than when injected subcutaneously. As a matter of fact, in injecting saliva or culture fluids subcutaneously I have observed that the animal commonly presents no indications of being ill during the first twenty-four hours, and that during this time the inflammatory œdema at the point of puncture has made but little progress, as shown by the slight extent of local tumefaction. The pleurisy and fibrinous exudation upon the surface of the lung, in these rabbits, corresponds with what the authors above referred to have reported as a result of the injection in the same way of pneumonic exudate. It may be that if death had not occurred at so early a period, the lungs would have become involved, and that these cases could have been placed beside those reported by Talamon, in which unmistakable pneumonia is said to have been produced by the injection into the thoracic cavity of rabbits, of pneumonic exudate, or of culture fluids. The superior pathogenic potency of the micrococcus in my experiment above reported, is shown by the brief period between the time of injection and the date of death. But, although this period was less than twenty-four hours, the blood of these rabbits, as well as the effused serum in the pleural cavity, contained the oval micrococcus in vast numbers.

I have been led to suspect, from the fact that while fibrinous pleurisy is a constant result in the experiments reported by Talamon and by Salvioli, fibrinous exudation in the lung tissue is only occasionally found, that perhaps the development of pneumonia depends upon the penetration of the lung by the point of the syringe, and the injection of the infectious material directly into the lung substance. I can easily understand how this result might occur, if the local effect of the presence of the micrococcus in the lung is the same as when it is introduced into the subcutaneous connective tissue, that is the infiltration of the tissue with an inflammatory exudate, which gives rise to the appearance which I have generally spoken of as an inflammatory œdema.

April 7th, 4 *P.M.*—Injected into two rabbits through walls of thorax, right side, by means of hypodermic syringe, a small quantity of pneumonic sputum, from a case in the practice of Dr. Mitchell. The sputum was collected with due care by my laboratory assistant Dr. A. C. Abbott. Both rabbits were found dead the following morning. There was considerable turbid serum in the right pleural cavity, and a fibrinous deposit upon the surface of the lung of same side. Lung floated in water and gave no evidence of being inflamed, but was stained of a reddish color. The spleens of these rabbits were but slightly enlarged.

Remarks.—I have frequently noticed that in cases so quickly fatal, tumefaction of the spleen is not so marked as in cases of longer duration. The blood of one of these rabbits contained the oval micrococcus in great abundance; blood drawn from the femoral vein of the other contained a small number of the same organism in pairs and chains of three or four. The turbid serum from the pleural cavity contained the microbe in great numbers.

Since the above was written the following experiment has been made:—

On the afternoon of April 9th Dr. Councilman, Assistant in Pathology in Johns Hopkins University, and Pathologist at Bay View Hospital, handed me a portion of hepatized lung from a case of pneumonia at Bay View, in which the post-mortem examination was made within six hours after death. The exudate, scraped from a freshly-cut surface of the lung tissue, was spread upon thin glass covers, and stained by the method recommended by Friedländer for the demonstration of his so-called pneumonia-coccus. Micrococci of oval form in pairs and in chains of three or four elements were found in abundance, but they were not surrounded by a capsule.

April 9th, 4 *P.M.*—A little of the exudate, scraped from a freshly cut section of the hepatized lung above referred to, was mixed with distilled water and injected into the right thoracic cavity of a small rabbit, by means of a hypodermic syringe. At the same time a little of my own saliva was injected into another small rabbit, of the same litter, in the same manner. Rabbit No. 1, injected with pneumonic exudate, was found dead on the morning of April 12. Upon opening the cavity of the thorax the lungs were found to present a normal appearance, except that the inferior lobe of right lung had a blood-red color, apparently due to a hemorrhagic infarct; both lungs floated in water. The right pleural cavity contained turbid serum, in which, as well as in the blood, the presence of numerous oval micrococci was demonstrated by microscopical examination. Several specimens were stained by the method recommended by Friedländer, but no distinct appearance of a capsule was made out. The spleen was considerably enlarged.

On the morning of April 13, rabbit No. 2 was found dead. There was but slight evidence of pleurisy, but the right lung was indurated, of a gray color, and sunk in water.

Remarks.—The spleen of this rabbit was not enlarged, and its blood did not contain the septic micrococcus. The development of pneumonia in this case was doubtless due to the fact that the point of the syringe had penetrated the lung, and the injection had been made directly into the lung tissue. Such a result must depend to a great extent upon accident, and in making injections through the thoracic walls, it is impossible to say in advance of the *post-mortem* examination whether the fluid has been injected into the pleural cavity or into the lung substance.

A microscopical examination of the lung after it had been hardened in alcohol showed that there was a necrotic focus of some extent surrounded by an extended area of indurated lung tissue, which presented the appearance of catarrhal rather than of croupous pneumonia. There was also interstitial hemorrhage in the vicinity of the necrotic centre, and there can be little doubt that this marked the point to which the syringe had penetrated when the injection was made. The oval micrococcus was not found in the inflamed lung tissue, and I am not able to ascribe to it any special import in the development of the inflammation in this particular case.

I must now refer to Friedländer's experiments upon mice, guinea-pigs, and dogs, and shall quote from the recent work of Prof. Sée already referred to:—

"All of the mice succumbed in from eighteen to twenty-eight hours after the injection. At the autopsy the lungs were found to be highly congested, almost empty of air, and to contain foci—*noyaux*—of red induration. Out of eleven guinea-pigs, six died with pneumonic lesions. Out of six dogs, one only succumbed. In another series of experiments mice were made to inhale a spray of a culture fluid containing the coccus in suspension. Several of these animals succumbed. At the autopsy Friedländer says that he found the appearance of typical pneumonia, and verified the presence of numerous micrococci with capsules in the lungs, the spleen, the blood, and the pleural liquid." (Vide ante, p. 54.)

I would like to inquire how it is that to produce pneumonia in animals, Friedländer and his followers consider it necessary to introduce the infectious material into the pleural cavity or into the substance of the lung itself with a sharp-pointed instrument. If this coccus is the germ of infectious pneumonia, one would think that to inject it subcutaneously ought to give rise to the local lesion, just as the subcutaneous injection of tuberculous sputum gives rise to tuberculosis of the lungs. We find from the accounts given that when injected into the lung or introduced by inhalation, the micrococcus invades the blood, and produces enlargement of the spleen; but to produce the characteristic local inflammation which it is supposed to be the especial function of this micrococcus to produce, it does not suffice to inject the micrococcus beneath the skin or into the blood, it must be injected through the walls of the thorax with a sharp-pointed syringe. And when so injected only a certain proportion of the animals have pneumonia. Prof. Sée, while accepting Talamon's experiments upon rabbits as demonstrating the infectious nature of croupous pneumonia is not inclined to accept the demonstration which Friedländer claims to have made by his experiments upon mice. He says:—

"The rather brief description of the lesions in the mouse, which Friedländer has given, leaves some doubt in the mind; for the presence of centres—*noyaux*—of induration in the congested lungs does not suffice to characterize fibrinous pneumonia."

I would here call attention to the fact referred to in my first paper on a fatal form of septicæmia in the rabbit, viz.: that in this infectious disease there are sometimes found hemorrhagic infarcts in the lungs. I quote from this paper as follows:—

"Pasteur says, 'The lungs are frequently filled with the noyaux of pulmonary apoplexy.' (I have found this to be the case in one out of three rabbits examined since my attention has been directed to this point.) 'A character more constant than the last (not more constant, however,

than that which relates to the volume and color of the ganglions), is the state of the trachea, which is almost invariably red, congested, with little hemorrhages from the smallest vessels.' (I have found a marked congestion of the vessels of the trachea in the three cases in which I have examined it, and in one case the lymphatic glands of the axillæ were enlarged and congested.)"

The possibility has frequently occurred to me, that this same micrococcus may be concerned in the etiology of diphtheria. The fact that it causes inflammatory œdema of the subcutaneous connective tissue, and that when introduced into the pleural cavity it causes a copious fibrinous deposit upon the lung, taken in connection with the fact that it has been shown to be a pathogenic organism having different degrees of potency under different circumstances, seems to justify the suspicion that there may be a pathogenic variety which has a special propensity for locating itself in the mucous membrane of the fauces. Certainly the suddenly developed inflammatory œdema and fibrinous exudation which characterizes malignant diphtheria, corresponds with the local effect produced when this micrococcus is inoculated into susceptible lower animals. The experiments of Löffler[1] seem to show that the widely distributed spherical micrococcus which forms long chains and zoöglœa masses is not the germ of diphtheria, and this observer speaks of a bacillus as being found in the deeper portion of the pseudo-membrane in cases of diphtheria. I have no exact information as to the morphology of this bacillus, but knowing that in the nomenclature of some bacteriologists, the name micrococcus is reserved exclusively for spherical organism, I am not prepared to dismiss this supposition without further investigation.

I herewith submit the following conclusions for consideration:

The pneumonia-coccus of Friedländer is identical specifically with the micrococcus previously described by me, and which is commonly found in normal human saliva. The capsule, or mucous envelope, which sometimes surrounds this micrococcus, described by Friedländer in 1883, and photographed by me two years previously, cannot be accepted as a distinguishing character of this species, inasmuch as it is not constantly present, and the circumstances upon which its development depends, have not been accurately determined. It is established that this is a pathogenic organism, so far as certain lower animals are concerned, and that its pathogenic power varies under different circumstances. It seems extremely probable that this micrococcus is concerned in the etiology of croupous pneumonia, and that the infectious nature of this disease is due to its presence in the fibrinous exudate into the pulmonary alveoli.

But this cannot be considered as definitely established by the experiments which have thus far been made upon lower animals. The constant presence of this micrococcus in the buccal secretions of healthy persons indicates that some other factor is required for the development of an attack of pneumonia; and it seems probable that this other factor acts by reducing the vital resisting power of the pulmonary tissues, and thus making them vulnerable to the attacks of the microbe. This supposition enables us to account for the development of the numerous cases of pneumonia which cannot be traced to infection from without. The germ being always present, auto-infection is liable to occur when from alcohol-

[1] I have not the paper of this author at hand, and am indebted to an editorial in the *Medical Record* of Jan. 31, for the facts here given.

ism, sewer-gas poisoning, crowd-poisoning, or any other depressing agency, the vitality of the tissues is reduced below the resisting point. We may suppose also that a reflex vaso-motor paralysis, affecting a single lobe of the lung, for example, and induced by exposure to cold, may so reduce the resisting power of the pulmonary tissue, as to permit this micrococcus to produce its characteristic effects.

Again, we may suppose that a person whose vital resisting power is reduced by any of the causes mentioned, may be attacked by pneumonia from external infection with material containing a pathogenic variety of this micrococcus having a potency, permanent or acquired, greater than that possessed by the same organism in normal buccal secretions.

The figures which illustrate this paper are from the following sources:—

Figures 1, 2, 3, and 4 from camera lucida drawings made by my laboratory assistant, Dr. A. C. Abbott, of Baltimore, to whom I am also indebted for valuable assistance in conducting the recent experiments herein recorded. The drawings were made with an amplification of 2,500 diameters, obtained by the use of a Zeiss's one eighteenth inch homogeneous oil immersion objective and a high eye-piece (Powell and Lealand large stand, tube drawn one inch). These drawings have been reduced by photography to two-fifths, in order to give an amplification of 1,000 diameters in figures.

Supplementary Remarks.

The above paper was read before the Pathological Society of Philadelphia on the 17th of April and published in the July number of the American Journal of Medical Sciences for the same year. Unfortunately the writer was absent in Europe when this paper was published, and failed to receive the proof which was sent to him for correction, This failure has resulted in a grave error through which the amplification of Figs. 1, 2, 3, and 4 is 2,500 diameters, while the descriptive text gives it as 1,000 diameters. As stated in the last paragraph of the paper, the figures were drawn under the camera lucida with an amplification of 2,500 diameters. They were to be reduced by photography to 1,000 diameters. *But the reduction was inadvertently not made,* and the comparison of these figures with Fig. 6, in which the amplification is 1,000 diameters, shows a very great difference in the dimensions of the oval micrococci from different sources, which might be taken as evidence of a specific difference, instead of specific identity as claimed, if it were not due to a mistake. The cuts which have been prepared for the present volume are better, but a comparison of Fig. 6 with the photo-micrograph from which it is copied shows how difficultit is for the wood-engraver to reproduce faithfully a photograph from nature.

In claiming that the pneumonia-coccus of Friedländer is specifically identical with my *Micrococcus Pasteuri,* it must be understood that I use the word *species* in its botanical sense, and that I recognize different varieties—physiological or pathological varieties of more or less permanence —of the same species.

It may be well to recapitulate briefly the principal evidence which has led me to the conclusions stated.

Note.—As this book is going to press, a letter from Prof. Sée has the following reference to the above article: "Dr. Sternberg n'est pas si éloigné de mon opinion, qu'il ne connait pas encore. ni des expériences de Talamon."—Trans.

a. My own observations and the figures given by Talamon and Salvioli have convinced me of the morphological identity of the oval micrococcus found in pneumonic exudate and my *M. Pasteuri*, as found in the blood of a rabbit killed by the injection of human saliva. The mode of grouping, in pairs, or in chains of three or four elements, and the occasional appearance of a mucinous envelope (capsule of Friedländer) which is developed by a special method of treatment (staining with iodine solution) completes the evidence of specific identity from a morphological point of view.

b. Injections of pneumonic exudate and of liquid cultures from the same, made by Talamon in France, and by Salvioli in Italy, into the pulmonary tissue of rabbits, have, in a majority of the cases reported, caused the death of the animal, and it is noted that the blood contains the oval micrococcus in abundance, also (by Salvioli) that the spleen is enlarged, and that an infectious disease is induced. The accounts given by both of these authors, induce me to believe that this infectious disease induced by injecting pneumonic exudate into the pleural cavity or pulmonary tissue of rabbits, is identical with the fatal form of septicæmia in rabbits, previously described by me, which is induced by the subcutaneous injection of human saliva.

c. In my own injections of pneumonic sputum, made subcutaneously, and of exudate scraped from a freshly cut section of a hepatized lung (six hours after death), I have obtained results identical with those obtained by injecting my own saliva into rabbits.[1]

d. Injections of cultures of *M. Pasteuri* into the pleural cavity of rabbits, induces a fibrinous pleurisy, as is the case when pneumonic exudate is injected in the same way, and in both cases the blood is quickly invaded by the oval micrococcus, and death occurs from septicæmia.

e. The experiments of Talamon and of Salvioli show that the production of fibrinous pneumonia is not a constant result of injections through the thoracic walls of pneumonic exudate, or of cultures from the same. When it occurs, it appears probable that it is due to the fact that the point of the syringe has penetrated the pulmonary tissue, and the injection has been deposited in the substance of the lung. This supposition is supported by the writer's experiment, in which pneumonia resulted from an injection of a gelatine culture of *M. Pasteuri* directly into the substance of the lung.

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[1] It is not my fault that these experiments have not been more numerous. I have done my best to obtain material in Baltimore, but not being connected with a hospital, and not having a private practice, I have been obliged to rely upon other physicians for assistance in obtaining pneumonic material for experimental purposes and have been greatly disappointed in not securing a greater number of satisfactory samples during the season when pneumonia was most prevalent in this city.

APPENDIX B.

ON BACTERIA.[1]

BY

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MEMBER OF THE ACADEMY OF MEDICINE AND OF THE COUNCIL OF HYGIENE AND OF SALUBRITY OF THE SEINE. PHYSICIAN TO THE HOSPITAL COCHIN, PARIS, FRANCE.

Bacteria, and by this word we mean all the microbes or figured ferments, are certain minute living forms, placed on the confines of the vegetal and animal kingdoms. There is, however, at the present day a general agreement in regarding them as the smallest known vegetal organisms.

Observed as early as the end of the seventeenth century by Leuwenhoeck, then described in many of their forms by Müller in the eighteenth century, the bacteria were classed by Ehrenberg in 1830 in the family of *vibriones*, and made the subject of more precise investigation, although considered at first as infusoria or animalcules.[2]

Cohn was the first in 1853 to range them among the plants. Nägeli later classed them among the Fungi, of which he made three distinct and independent groups—the Moulds, the Saccharomycetes, and the Schizomycetes (Bacteria, Vibriones), and in this classification Nägeli is in accord with Barry.

More recently Cohn, taking for his starting point this view that the Schizomycetes resemble the Algæ more than the Fungi, has suggested for them the name of *Schizophytes*, instead of the *Schizomycetes*, which the Professor of Breslau had given them.[3] As for the division of *Schizomycetes*, organisms which alone ought to occupy us here as being the agents of morbid fermentations, their natural history is too imperfect to warrant us in attempting an exact classification.

These micro-organisms are almost unknown in their morphological constitution; they are only known and characterized in respect to their physiological effects. Moreover this question of the physiological action of the microbes is still involved in great darkness and perplexity.

Some, as Klebs, pretend that the cell progeny always resembles the parent cell; others, as Cohn, claim that a given species may engender

[1] In this article the additions by the translator are enclosed in brackets.
[2] Pasteur, Acad. des sc., 1863, t. lvi., 420, 1190; and t. lii., 1861, p. 344.
[3] Cohn, Treatise on the Biology of Plants, 1875.

divers states and forms which are only phases of development, varying with the environment. Again, we shall be obliged to consider these microorganisms less from the point of view of morphology, than from that of the rôle which they play in the nature and in the production of diseases.

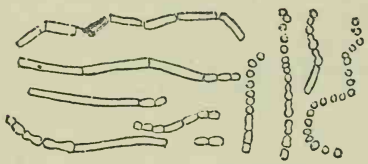

FIG. 1.—Bacilli in course of segmentation. × 1000.

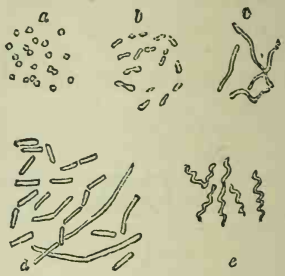

FIG. 2.—Schizomycetes.

However, for a proper comprehension of the subject, we have need of a classification, and that is still the most satisfactory which is based on morphological distinctions. Hence it is that the following provisional arrangement has been adopted and which we present as the morphological classification which is most in accordance with actual knowledge: 1. Microbes having the form of *points* (Moner, Monad, Micrococcus), which are immobile and have been considered as the spores of other microbes. 2. The linear immobile microbes (Bacteridia and Bacillus). 3. Mobile cylindrical microbes with rounded ends or constricted in the middle in the form of a figure 8 (Bacteriens). 4. Mobile flexuous microbes, which are undulating or eel-shaped (Vibriones). 5. Mobile spiral microbes having the aspect of a corkscrew (Spirillum and Spirochetæ). 6. Capitated microbes. Mobile rods having one extremity, or sometimes both, with a globule longer and more refractive than the rest of the body, and which has been considered as a spore on the point of being detached (Bacterium capitatum).

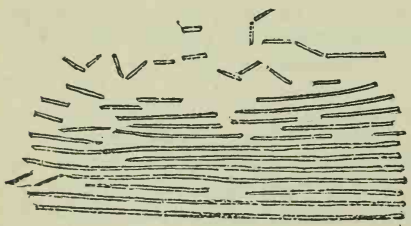

FIG. 3.—Bacillus Subtilis, formed at the surface of an infusion of boiled hay, after 25 to 48 hours. × 650.

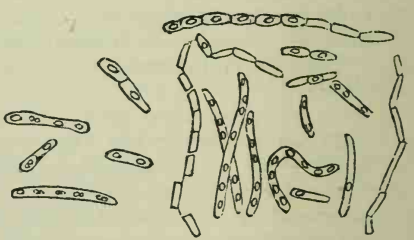

FIG. 4.—Phases of development of a bacillus of more than the ordinary dimensions. × 1600.

Besides these six states, the microbes form colonies which change the aspect of the elementary cells. 1. Bacteria agglomerated in microscopic masses, surrounded by a sort of jelly, which causes them to cohere, and renders them immobile—(Zoöglœa.) 2. Bacteria in sheets (*en nappe*) and immobile—(Mycoderma). 3. Bacteria clustered end to end—(Leptothrix). 4. Micrococcus forming a chaplet or necklace—(*Torula*).

It is necessary to fix these terms and dispositions well in the memory in order to understand the subject and intelligently to *see* with the microscope.

Another classification has been assigned to these infinitely little organisms, which is of a physiological kind, *i.e.*, having reference to their action on organic or living matter. Thus it is, that in accordance with the old adage: "it is at the foot of the wall that you detect the mason," mycologists have recognized a putrid bacterium, a septic vibrio, lactic, butyric, gallic, alcoholic, acetic ferments, etc.

Of these bacteria, some cannot live except by assimilating free oxygen (*aërobic* microbes), and are not *ferments;* others can derive the means of subsistence and thrive apart from free oxygen, by wresting this vital air from its combinations, notably from fermentative matter, which always contains oxygen in abundance; these *anaërobic* organisms are ferments in these conditions. It is this that has led Pasteur to say that "fermentation is life without air." [1]

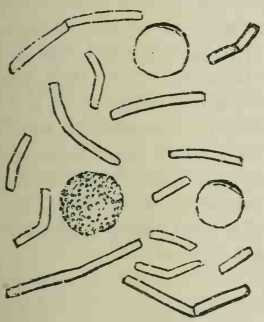

FIG. 5.—Bacilli of a size larger than the average ×1000.

FIG. 6.—Growth in filaments of the bacilli of the adjoining figure (Fig. 5).

The schizomycetes almost all possess spontaneous movements, ordinarily rapid, which Cohn and Koch attribute to certain vibratile cilia. Even the bacteridium of Davaine seems to have motion, contrarily to what has been before believed. (Ewart.)

These bacteria multiply with great rapidity by transverse fission. Each cell divides into two, which sometimes live end to end for a considerable time, forming jointed rods which are often cemented at the articulations.

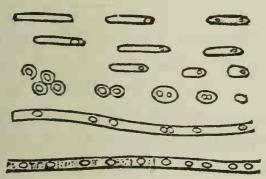

FIG. 7.—Filaments producing spores, and spores transforming themselves into rods (Lewis).

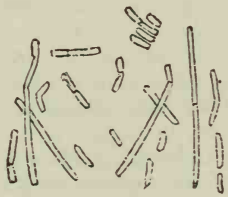

FIG. 8.—Phases of development of a bacillus (Lewis).

Koch and Pasteur have moreover described in these microphytes a kind of reproduction by asexual spores. These spores or "corpuscle germs" may remain a long time at rest and undergo numerous changes without losing their faculty of germination. As they seem only to form in bacteria when placed in unfavorable conditions, they ensure the perpetuation

[1] Pasteur, Acad. des Sc., Compt. Rend., Jan. 13, 1879, p. 58.

of these dangerous little organisms. According to Pasteur, these germs resist the action of absolute alcohol, boiling water, and compressed oxygen. We shall see later what must be thought of these affirmations. According to this authority, the "corpuscle germs" can hardly be called *living;* they have none of the characters of life, that is to say, nutrition, development, and generation; it is a latent life, a potential activity.

Generally the bacteria are killed by a temperature of 144° to 160° F., but certain of them resist prolonged temperatures of from 212° to 230° F. (Eidam, Cohn, and Pasteur.)

In this state, according to Pasteur, they speedily undergo transformation from a condition of relatively fragile cells into that of spores or corpuscle germs, and then brave the greatest vicissitudes. This is an opinion which we shall discuss farther on.

The bacteria do not live in all media; there are some which, while undergoing development in the blood of hares, sheep, guinea-pigs, and rats, cannot live in that of a dog or in that of birds. (Of this we have an instance in the *bacillus anthracis.*) So also the vibrio ferment which destroys *dextro tartaric* acid has no action on *lævo tartaric* acid; that is to say, does not develop in a medium where the latter exists to the exclusion of the first. *Racemic* acid offers the singular combination of one molecule of *dextro tartaric* acid, with a molecule of *lævo tartaric* acid; now if the racemate of ammonia be submitted to the fermentative action of the *dextro tartaric* bacterium, the process continues till this acid has totally disappeared, while the *lævo tartaric* acid remains intact.

FIG. 9.—Formation of spores in certain rods (Lewis).

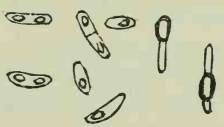

FIG. 10.—Isolated spores undergoing germination (Lewis).

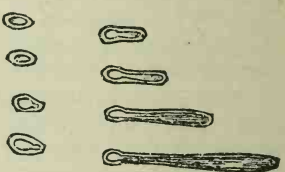

FIG. 11.—Spores developing into rods (Ewart).

These examples suffice to show that certain media of a given composition are necessary for the life of given bacteria; we shall later on see the importance of this fact.

Active agents of nearly all the fermentations, these infinitely little beings, whose mean diameter varies from that of a red blood globule to double this diameter (6μ. to 12μ.), are according to Pasteur's school the cause of all the infectious diseases. Their pathogenetic rôle we shall better understand when we know the part which certain of these microbes have in the decomposition of non-living organic substances.

FERMENTS AND FERMENTATION.

The rôle of the infinitely little (ferments) in nature is considerable. It suffices to recall to mind the conditions of existence of the larger members of the vegetable kingdom, to see that their life, and consequently that of the animals which live on them, is nothing but the synthetical outcome, the organization, of gases borrowed from the atmosphere, or saline or azotized matters existing in solution in the water. The vegetables utilize

for their life and development, the air, the water, and the salts dissolved in it, the organic matters contained in the soil, and with these elements form cellulose, starch, sugar, gum, oils, and albuminoid matters. But when once produced, this organic matter becomes solid and insoluble in water. It loses its former motion, and is no longer fit to nourish a new vegetal, and unless by some transformation it is made to re-enter the *general whirlpool* (*tourbillon général*) under gaseous or liquid form, the atmosphere, once exhausted of its carbonic acid and its oxygen, the soil deprived of its organizable saline or organic materials, would be unable longer to support life. The earth, never again to be inhabited by living things, would become a tomb rolling in boundless space.

The chemical statistics of organized beings, for which we are indebted to Dumas and Boussingault, have enabled us to comprehend the composition and reciprocal relations of animals and vegetables. In these tables we see as on a balance sheet the mutual exchanges of the elements, and obtain a clear conception of that grand phenomenon which Moleschott has so well described in his "Circulation of Matter."

But dead organic matter is not slow to return to the air and water by putrefaction, and the principal agents of this phenomenon are the microbes. Apart from their action, organic matter but tardily undergoes destruction under the slow combustion produced by the oxygen of the air. By the help of the microbe ferments, however, the destruction becomes rapid and complete.

They are very small, nevertheless, these microbes! Yet while the large animals, which themselves also are only burners and destroyers of matter, are able to transform each day but a small fraction of their weight, the ferments, on the contrary, metamorphose quantities a thousand times larger than themselves.

One scruple of yeast will transform in a day fifteen times its weight of sugar. The *Mycoderma Aceti* converts daily into vinegar (acetic acid), many hundred times its weight of alcohol, and dies sometimes under the action of the relatively elevated heat which is developed by the oxidation which it provokes.

The *Aspergillus Niger* metamorphoses in the gallic acid fermentation, two thousand times its own weight of tannin. (Van Tieghem.) You see, then, what prodigious activity the ferments have. This activity the microorganisms also employ, unhappily, in attacking living beings, without even sparing man, and a number of diseases are of parasitic nature. It becomes important, then, to determine if these formidable parasites may be generated "*de novo*" within our tissues or organs, by a transformation of the living matter, or if, on the contrary, they always take their origin from antecedent living organisms like themselves. The word *contagion* would have a quite different meaning in the two cases, and the means of self-preservation from, or warfare against, contagion would not less differ. This involves the consideration of the subject of Heterogenesis, which we shall presently take up.

Pasteur admits two kinds of fermentation; the one direct or caused by living agents, the other indirect, produced by soluble ferments. Now, if we take for example of fermentation, that which is excited in saccharine liquids under the influence of the yeast plant (*Saccharomyces cerevisiæ*) we see that the change which is apparent, consists in the disappearance of the sugar and its replacement by alcohol, carbonic acid plus a certain quantity of glycerine, succinic acid, etc. We

notice that chemical phenomena and the presence of *saccharomyces cerevisiæ* are correlative; but, as Schutzenberger says ("Fermentations," in the International Scientific Series, 1878): "as for a more precise relationship between the chemical phenomenon and the physiological functions of the organism-ferment, it remains still to be discovered, and all that has been said, described, or advanced, to solve the question, lacks experimental control."

Among the indirect fermentations, defined by Schutzenberger as "reactions whose cause is derived from, but may act apart from an organism," we may take the following example: when you add pancreatic juice to fat, there is first emulsification, then saponification, that is to say breaking up by hydration into a fatty acid and into glycerine. Here you have a physiological fermentation. But no more than in the preceding case, are we able to give an explanation. It is the same with the transformation of starch into grape sugar under the action of ptyalin, and with the transformation of albuminoids into peptones under the influence of gastric juice; the intimate mechanism of the metamorphosis is unknown to us. The two kinds of fermentation have this in common, viz., that a little quantity of ferment may act on an enormous mass of fermentable matter and transform it. The difference between the two is not then perhaps quite as well defined as Pasteur would have it. The only difference is that in the case of direct fermentations, the living organism may always be found, while in the indirect fermentations, the living being which has produced the fermentation may not be present at the moment when the ferment acts, the latter being soluble, the former insoluble.

But the direct fermentations are often preceded or accompanied by indirect fermentations, whose inception is due to the direct ferment, and on the other hand, the chemical phenomena characteristic of this or that direct or indirect fermentation may be determined by chemical agents of mineral origin.

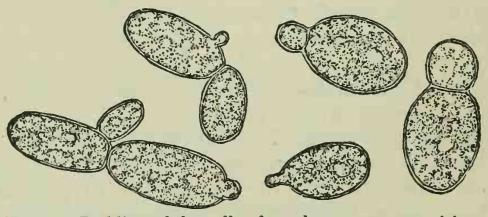

Fig. 12.—Budding of the cells of saccharomyces cerevisiæ.

Thus sugar undergoes decomposition before the production of alcohol in the alcoholic fermentation. Berthelot has shown that before fermentation, cane sugar undergoes a hydration and breaks up into two opposite glucoses; grape sugar, which causes the plane of polarization to turn to the right, and lævulose, or uncrystallizable sugar, which causes it to turn to the left, and this savant has demonstrated that this decomposition is produced by an indirect soluble ferment which is excreted by the cells of the *saccharomyces cerevisiæ*, and which may be found in the filtered water in which yeast has been washed. The same ferment produces, then, the two sorts of fermentation: 1. the breaking up of the cane sugar into two other sugars; 2. fermentation, or transformation of these two sorts of sugars into alcohol and carbonic acid. But this is not all. These fermentations may be provoked by chemical agents purely mineral. The splitting up

of cane sugar, of which we have just spoken, is brought about quite as well under the action of acids as under the influence of yeast; even by the mechanical act of trituration a part of the sugar undergoes conversion. The saponification of fats which the pancreatic ferment determines, the transformation of amylaceous matters into dextrine and glucose by the salivary diastase, are equally produced by boiling alkalies or acids.

The view of Pasteur that "the chemical act of fermentation is essentially a correlative phenomenon of a vital act, commencing and ending with the latter," is, then, much too absolute. What is true is that fermentations are but the consequence of an antecedent chemical action. Life being itself but a molecular movement of matter, may, like all the physico-chemical movements, cause fermentation. It is only because he has allowed himself to be carried away by his vitalist, metaphysical or religious notions, which he took the opportunity recently to avow systematically, that Pasteur has been led to make of fermentations phenomena of a special nature. We have seen this savant, beguiled into airy realms of speculation, called back to the true scientific philosophy by a philosophical historian, Rénan,

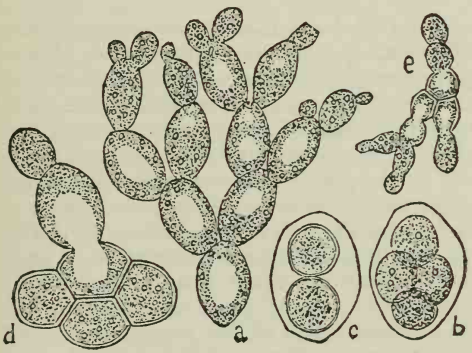

Fig. 13.—Cells of the saccharomyces cerevisiæ in full activity: *a*, *d*, *e*, budding; *b*, *c*, forming spores.

on the very occasion of the introduction of the former to the French Academy. (Reception of Pasteur to the French Academy, April, 1882.)

Charles Robin says: "Fermentation is a particular instance of the nutrition of divers vegetables of the uni-cellular or pauci-cellular state, and even of apples, or other fruits, at their evolutive period of maturation, and is but a kind of nutritive disassimilation with disengagement of heat."

The difference between the respective living ferments such as the alcoholic, tartaric, lactic, etc., are of just this character, that each, according to its own constitution, thrives on some particular nutritive ingredient, which it assimilates and disassimilates at will, while it dies in another medium not suited to it, but which is perfectly suited to some other ferment organism. Thus lactic acid forms a fermentable principle suitable to one kind of microbe, sugar a principle suitable to another, butyric acid a principle suitable to another, etc. The important fact to keep in mind, then, is that each fermentable solution, whether of glucose, tartrates, or urea, etc., has its particular micro-organism which thrives on it exclusively.

In like manner, the pathogenetic action of the schizomycetes is limited to this power of organic disintegration, in which they act the part of ferments in furtherance of their own nutrition.

But as an illustration of the fact that in these difficult questions, still so obscure, it will not do to hasten to conclusions, it is noteworthy that at the moment when Pasteur was establishing a capital distinction between the artificial organic products and the compounds formed under the influence of living beings,[1] Perkin and Von Dupa overthrew this distinction by transforming succinic acid into tartaric and para-tartaric acids. But the latter breaks up, as Pasteur has himself recognized, into dextro tartaric acid and into lævo tartaric acid, and Jungfleisch has shown that this last when heated in water up to 175°C. is converted into para-tartaric acid. Moreover he has obtained synthetically succinic acid by means of potassa and cyanide of ethylene. Now the vibrio ferment of the tartaric fermentation has no action on the lævo-tartaric acid, that is to say, it does not develop in this medium. Thus the barrier is broken down between the living chemistry and the chemistry of the laboratory. "If the living cells," says Schutzenberger, "provoke reactions which seem specific for them, it is because they realize conditions of molecular mechanics which we have not been able yet to seize, but which the future will enable us *without doubt* to find."

Liebig has explained the action of the saccharomycetes in the alcoholic fermentation by this theory, that the ferments are albuminoid substances which possess the property of being decomposed themselves, and of entailing the decomposition of other substances with which they find themselves in contact. Kingzett (Journ. of the Society of Arts, Mar. 1878) has formulated in excellent terms this physico-chemical theory or chemistry of contact. "Every mechanical or other movement, exercises an action on the force which determines the states of bodies. Thus a crystal of sulphate of soda, or simply a grain of sand, plunged into a saturated solution of sulphate of soda, may rapidly determine the formation of quantities of crystals of sulphate of soda. Likewise when you touch, however lightly, with a pen or glass rod, the fulminates of silver and mercury, they explode with violence. A better example still of this phenomenon, is furnished by the reaction which is produced when you bring together oxide of silver and peroxide of hydrogen; as a result of this mechanical union there is formed metallic silver and free oxygen; the peroxide of hydrogen, a body naturally very unstable, decomposes at the moment even of its formation, giving rise to water and oxygen; when this decomposition takes place in contact with oxide of silver, it determines in the latter body the same tendency to decomposition."

In fine, in order to show that the hypothesis of Liebig is but a deceptive mirage, Pasteur[2] composed certain artificial media consisting only of pure water with the mineral and ternary substances necessary for the life of the ferments, and the germs of the ferments of those divers matters. In these conditions the fermentations went on perfectly, every albuminoid matter being excluded, the ferment appeared like a living being borrowing from the fermentable matter all the carbon of its successive generations, and from the mineral matter, its nitrogen, phosphorus, potassium, magnesium elements, the assimilation of which is necessary to the nutrition of all organisms.

"In these conditions," adds Pasteur, "not only the theory of Liebig was shown to be without foundation, but the phenomena of fermentation

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[1] Leçons de la Société chimique de Paris, 1860.
[2] Comptes rends. Acad. des Sciences, Paris, 1880.

presented themselves, as simple phenomena of nutrition." And elsewhere he says: "Not only have I demonstrated that the ferments of the fermentations properly so called, are not dead albuminoid matters, but really living beings; I have, moreover, caused the fermentation of sugar, of lactic acid, of tartaric acid, of glycerine, etc., in media exclusively mineral; an incontestible proof that the decomposition of fermentable matter is correlative with the life of the ferment, being indispensable to its nutrition; under these circumstances, it is impossible that in the constitution of the ferments which take their origin in these media, there can be a single atom of carbon which is not taken from the fermentable matter."

Thus it is that, according to Pasteur, yeast determines the decomposition of sugar and the alcoholic fermentation by wresting from the sugar the oxygen necessary for its respiration. It acts here like the bacteria which provoke the tartaric and butyric fermentations, and which not only can live and multiply away from contact with free oxygen, but which perish and cease to excite fermentation if free oxygen is dissolved in the medium in which they live.

Nevertheless yeast lives in contact with air, and if under conditions where the air is excluded, it *seems* to respire at the expense of the sugar and take from the latter its oxygen, it is not the less true that in a liquid containing sugar and yeast exposed to contact with the air, and spread out, the production of alcohol and the multiplication of the yeast go on with a rapidity much greater than when the yeast solution is removed from the air. This seems in contradiction with Pasteur's theory, as De Lanessan remarks (Histoire Naturelle Medicale Botanique, p. 1293, Paris, 1880), for if the yeast decomposes sugar by respiring its oxygen, the alcoholic fermentation ought to be less rapid when the yeast finds at its disposal atmospheric oxygen in abundance, than when it is deprived of free oxygen and is compelled to obtain this element from the sugar exclusively.

In the discussion between Berthelot and Pasteur at the Academy of Sciences with reference to the posthumous note of Claude Bernard, the purport of which was that alcoholic fermentation is due to a soluble ferment, Berthelot vehemently contested the view of Pasteur in which the latter puts in opposition his well known theory of the aërobiotic microbes which consume free oxygen and the anaërobiotic microbes which consume combined oxygen. "Any such function," he says, "is purely hypothetical. Let us make this clear. If brewer's yeast takes from sugar its combined oxygen, one ought to be able to find in the liquids the deoxodized residuum, for example instead of C^{12} H^{12} O^{12} —C^{12} H^{12} O^{6}, or the products of its decomposition. What one does find in reality is alcohol and carbonic acid, the united weights of which represent almost exactly the weight of the sugar. No fact, then, warrants us in supposing that the ferments have the singular property of taking from the sugar a portion of its combined oxygen. At all events, it seems to me, as it did to Claude Bernard, that the tendency of science is to reduce the action of ferments to conditions purely chemical." This is in fact what has been successfully realized in the case of almost all the fermentations, as is proved by the history of the glucosic fermentation of starch in germinating barley, of fatty bodies in the intestine, of amygdalin in bitter almonds, of cane sugar undergoing inversion under the action of yeast, of urea in the urine, etc. A. Gautier has proved this fact anew for the pepsin ferments, by eliminating all possibility of life from the mycrozymes of Béchamp. (Acad. de Med., May, 1882.) This able chemist has proved

that the granulations of gastric juice,—Béchamp's mycrozymes—are not organized beings, for they are incapable of proliferating in the digestive media suitable for their activity, but previously sterilized by borax, phenol, hydrocyanic acid, and they easily digest albuminoid substances in presence of the most energetic poisons, such as prussic acid, which destroys all microbes. These insoluble corpuscles of pepsin slowly transform themselves into a digestive chemical ferment without organization and without life, and which may be obtained crystallized. (Acad. des Sciences, 1882.) Quite recently Darsonval has communicated to the Biological Society the result of an experiment which confirms the view of Claude Bernard, that the alcoholic fermentation is accomplished by the intervention of a soluble ferment, and not of a figured ferment.

Into a cast iron tube, able to support a pressure of one hundred atmospheres, Darsonval poured sweetened water and yeast; then before closing it, he introduced a manometre. At the moment when this instrument marked a pressure of twenty atmospheres, the cork flew out. This fermentation could not certainly have been provoked by the life of a cellular organism, since, as Paul Bert has shown, carbonic acid under pressure is a *universal poison;* five per cent. of CO^2 in the air arrests germination. Can yeast be an exception to this biological law?

The respiration of living beings is always an accompaniment of nutrition; by this combustion, this oxidation, which incessantly goes on under the influence of the oxygen introduced into the organism by respiration, certain immediate principles undergo disassimilation, others are formed out of alimentary substances furnished to the economy by the surrounding medium. The action of the yeasts illustrates this general law, and numerous experiments have shown that sugar constitutes for them an excellent aliment. It would not, then, be difficult to defend the position that the alcoholic fermentation is rather the consequence of the *nutrition* of the yeast than of its *respiration.* In adopting this view, Schutzenberger and Monoyer arrive at this conclusion, that the yeast does not become a ferment because it respires a part of the oxygen of the sugar, but it may respire a part of this combined oxygen, and then multiply precisely because it produces disposable (*i.e.* utilizable) oxygen in decomposing the sugar for its sustenance.

"In examining the question from a more general point of view," adds Schutzenberger, "one may still say that the respiratory combustion is for the living being a source of energy necessary for its development. Now in the decomposition of sugar, there will be, according to the calculations of Berthelot, disengagement of caloric; the quantity of heat set free in this act would be about one fifteenth of the caloric disengaged by the complete combustion of the decomposed sugar. In this reckoning, account is not made of the heat which becomes latent in the dissolved sugar, nor of that which disappears with the solution of the alcohol formed; positive quantities which tend to elevate the figure of the caloric of fermentation. It is not, then, at all necessary to have recourse to the hypothesis of a combustion at the expense of the oxygen of the sugar, in order to explain how the phenomenon of fermentation may supply the combustion, and become a source of the energy indispensable to the development of the vegetal."

Béchamp has equally attributed to a phenomenon of nutrition the alcoholic fermentation caused by the saccharomyces in a liquid holding sugar in solution. The yeast nourishes itself with sugar and then excretes as

products of disassimilation, alcohol, carbonic acid, succinic acid, glycerin and tartaric acid.

Brefeld (in Rev. Inst. des Sciences, t. ii. 1878, pp. 538 and 563) has put forth an opinion similar to that of Béchamp. According to his view, yeast cannot live normally without free oxygen and, contrarily to the opinion of Pasteur, it never respires the oxygen which enters into the chemical constitution of the sugar. Fermentation, according to this experimenter, is due to this, that the yeast, after having assimilated the sugar of the nutritive liquid, excretes the carbonic acid and alcohol resulting from the decomposition of the sugar effected in the cells themselves. By reason of its great affinity for oxygen, etc., the yeast rapidly absorbs the free oxygen of the nutritive liquids, and when once this gas has disappeared, the growth of the torula is arrested; it sickens in these abnormal conditions of life which it creates; the fermentation which then results is nothing but a pathological effect.

According to Traube, fermentation is not necessarily a phenomenon correlative with the life of the cells. He bases his affirmation on these facts: 1. The embryonal cells of the yeast cannot develop without free oxygen: 2. The budding yeast, on the contrary, may multiply in a medium (like grape juice) without free oxygen; 3. If you deprive the liquid of air, the multiplication of the yeast soon ceases, even when there is a portion of the sugar remaining which is not yet decomposed, a fact which militates against the view of Pasteur, according to which the yeast takes from the sugar the oxygen necessary for its development and multiplication; 4. In a simple solution of sugar, the yeast provokes fermentation, in the absence of all trace of oxygen, but without undergoing any increase, which weakens the theory that fermentation is dependent on the respiration of the yeast plant; 5. The seeds of grapes may convert the sugar of the latter into alcohol even in the absence of air, when the juice is not able of itself to undergo fermentation.

Meyer has on his part enunciated a view which resembles that of Pasteur. He admits that certain lower organisms may live for a while without free oxygen and continue to grow, but this does not long continue. Thus he considers the respiration accomplished through the combined oxygen of the sugar as insufficient long to maintain the functions of the *saccharomyces cerevisiæ*, and thinks that the fermentation may be regarded as supplying the place of respiration in free oxygen, basing himself on this fact that the fermentation produced by yeast is the more rapid the more the fungus is deprived of free oxygen.

In fine, Berthelot (Chemical Synthesis) enunciates an opinion respecting fermentations somewhat like that of Liebig, when he says that "the causes which provoke fermentations seem analogous to those which give rise to actions of contact;" adding that fermentations provoke chemical reactions accompanied with the development of heat, a phenomenon which is not the outcome of the proper energy of the ferment, but which it provokes, perhaps, after the manner of agents of contact and by means of the formation of some intermediate compound. This does not hinder this savant from adopting, farther on, Pasteur's two groups of fermentations, while adding in another place this remark: that "chemical affinity, heat, light, and electricity, suffice to cause the elements to combine in organic compounds. In this way we produce an infinity of natural principles. The chemical effects of life are due to the play of ordinary chemical forces, just as the physical and mechanical effects of life take place according to

the play of forces purely physical and mechanical. In both cases the molecular forces put in operation are the same, for they give rise to the same effects."

Such are the principal rather contradictory opinions which have been put forth to explain alcoholic fermentation. While some say: "Wherever there is fermentation there is organization, development and multiplication of the cells of the ferment itself," others pretend that the affair is not so clear, and that there may be fermentation without life. While some distinguished observers refuse to the little plant discovered by Schwann and Cagniard Latour the property of living without free oxygen, others accord to it this faculty, and it is on this latter that is based Pasteur's theory, for the first time formally enunciated by Turpin; "No fermentation without the physiological act of vegetation," as well as the hypothesis of aërobiotic and anaërobiotic organisms. Till this question shall have been solved by indisputable experiments, it will be impossible to accept definitively Pasteur's theory.

In fine, what we know incontestably is that the *saccharomyces cerevisiæ*, placed in a solution of sugar from which the oxygen of the air is excluded, or even in contact with this gas, determines the decomposition of sugar into alcohol, carbonic acid, etc., a phenomenon which has received the name of fermentation; what we also know indisputably, is that the *saccharomyces* borrows from the solution, and in particular from the sugar, the materials necessary for its nutrition and multiplication, for it is easy to assure ourselves that under favorable conditions the quantity of yeast augments in proportion as alcohol is formed; we are then absolutely certain that the decomposition of the sugar which is produced under the influence of this cryptogam, is a phenomenon concomitant with the life of the fungus; and as life itself is but a succession of physical and chemical phenomena, we cannot doubt that the alcoholic fermentation is equally a phenomenon of physical and chemical order; but we are ignorant of the way in which this phenomenon is produced and what is its intimate nature. Does the yeast act by respiring the oxygen of the sugar, as Pasteur thinks and as Meyer admits? Does it act by simple action of contact? And is the phenomenon purely physical, as Berzelius thinks? Does it determine the decomposition of sugar, in itself undergoing decomposition, as Liebig teaches? Does it nourish itself on the sugar, to excrete forthwith, as products of disassimilation, alcohol, carbonic acid, etc., as Béchamp supposes? Or, lastly, does the yeast elaborate a soluble ferment which it then excretes, and which acts on the sugar like the diastase of saliva on starch, according to Berthelot's view? Which of all these hypotheses is the most probable? We are still ignorant.

If we have devoted so much consideration to the theory of fermentation, it is because at each moment we shall be obliged to make use of this word in the study of the rôle played by the microbes in the production of infectious diseases; and this is why it is desirable to fix well in our minds the full meaning of this word, for, key-stone of the arch of an entire doctrine, the latter will fall to the ground if it be demonstrated that fermentation is *not* life without air, just as spontaneous generation is overthrown, if it can be proved that no bacterium has ever originated in a putrescible infusion carefully protected from the germs of the inferior vegetal organisms floating about in the air.

To this question of microbes, then, are linked all the theories of heterogenesis and of panspermism, the latter opposing the theory of germs to

the theory of spontaneous generation. We cannot do otherwise than say something concerning the origin of these little beings which are soon to occupy our attention as factors of grave disorders in living organisms.

Heterogenesis and Panspermism.

Can any being whatever be born without parents? Antiquity answered yes. Aristotle maintained that animals are sometimes formed in putrefying soil; he supposes that eels come from the mud of rivers by fermentation, and caterpillars from dew in a similar manner. Virgil's directions for the production of bees from the putrefying entrails of a stag are known to all readers of the Georgics. Sampson's riddle in Judges xiv. 14, assumes the generation of a swarm of bees from a carcass of a lion. Nearer our own times (1577–1644), Van Helmont gives special instructions for the artificial production of mice. Cardan asserts that water engenders fishes, and that many animals spring from fermentation, and Kircher, in his "Mundus Subterraneus" describes and actually figures certain animals which were produced under his own eyes by the transforming influence of water on the fragments of the stems of different plants.

In the seventeenth century Redi, then Swammerdam, and Vallisneri, and Reaumur showed that the maggots which cover putrefying meat did not come into being spontaneously, out of the midst of the rottenness, as was supposed, but that they are derived from eggs deposited by flies, and that it suffices to keep the meat in a receptacle well covered with gauze to prevent the maggots from making their appearance. Spontaneous generation then received a severe shock; the microscope was destined soon to revive it for a while.

Make an infusion of hay, of brewers' yeast, or of meat; that is to say, allow these vegetable or animal organic substances to digest in water a few hours, then filter. You will thus obtain clear liquids, and the microscope will not detect any microbes.

Place these infusions in a room at a temperature of from 96° to 104° F., and after twenty-four or forty-eight hours they will have become turbid and their surface will be covered with a gelatinous layer. A marked alteration will have taken place.

Take now one drop of one of these liquids and examine it by a magnifying power of 400 to 500 diameters. A surprising spectacle presents itself to our eyes: the field of the microscope is filled by living beings, some under the form of long flexible filaments undulating like eels—these are *vibriones;* then simple or articulated rods, very short and mobile—these are *bacteria;* others present themselves under the form of thin straight or jointed rods, mobile or immobile, in which each joint remains rigid—these are *bacilli;* in fine, there are some which are formed by rounded or oval cells, isolated or grouped in 2, 4, 8, forming thus chains resembling chaplets—these are *micrococci.*

There are even organisms higher in the scale of organization, monads, furnished with a vibratile tail, infusoria, like the kolpodes, amœbæ, etc.

It was in this world of the infinitely little that the advocates of heterogenesis now took refuge, following in the footsteps of Buffon, who affirmed the existence of *organic molecules,* and of Needham, who made his celebrated experiments in 1745. Needham introduced putrescible substances into flasks which then were hermetically sealed and heated to ebullition. "If there were any germs in these flasks," he argued, "the heat must infal-

libly have killed them; if then we should find living organisms in these liquids afterwards, they could have come into existence only by spontaneous generation." This view, based on such seemingly conclusive experiments, was long accepted, but met in 1765 an able opponent in Spallanzani. He, repeating the experiments of Needham, but heating the closed flasks much longer than the latter, suppressed all production of infusoria.

"If your infusions remain sterile," responded Needham, "it is because you heat them too much; you thus alter the air of your vessel or you annihilate the vegetative force of your liquids."

Gay Lussac brought his great authority to the support of Needham. This skillful physicist introduced into the top of a graduated bent tube, filled with mercury, several bunches of grapes, whose surface he washed several times with hydrogen gas in order to remove the last traces of air remaining adherent to the pellicles; then he crushed them *in situ* by means of a bent rod, introduced through the mercury. No fermentation was produced; he then caused a little oxygen to be introduced in contact with the must, and shortly after the fermentation commenced. Hence he concluded that oxygen is necessary for the commencement of fermentation. Schwann weakened the force of this experiment.

Meat broth remained intact in a flask where it had been previously boiled, and where, after cooling, a continuous current of air was maintained, precaution being first taken thoroughly to calcine the air by passing it through a tube heated to redness. This took away all the validity of Needham's objection relative to the impurity of the air. The experiments of Schultze, in 1837, who substituted for calcination of the air, its passage through sulphuric acid and potassa; of Schröder and Dusch, who showed that one could obtain the same result as Schwann by filtering the air through a cotton tampon, equally gave a death blow to the hypothetical objection of the vegetative force of the air. But always this difficulty remained: what was this principle which the fire destroyed and which the cotton arrested, and which gave to the infusion its fecundity? Things were in this condition when Pouchet, director of the museum of Rouens, announced to the Academy of Sciences, in 1858, that he had succeeded in experimentally demonstrating the reality of spontaneous generation.

Let us cite, among others, the two following experiments of Pouchet.

Pouchet introduces into a flask a decoction of hay or of nutgalls, of urine, of beer, of albumen, or of flour paste; he lets it boil several hours, and when he has acquired a certainty that the apparatus is at a temperature of 212° F., he closes it with a tight stopper, traversed by a wash tube containing sulphuric acid. He continues the ebullition during a certain time, then abandons it to the air. Microphytes, nevertheless, develop in this flask, although Joly and Musset have proved that air passed through sulphuric acid is completely sterilized.[1] The calcined air which contains the germs of generation, according to the panspermists, when it reaches the infusion thus sterilized, none the less provokes the genesis of bacteria.

The apparatus which Pouchet employed to establish this fact, consisted of a flask with a long neck provided with a stop cock. This communicated

[1] G. Pennetier, Les Origines de la Vie, Paris, 1868, p. 207. [Tyndall, however, has shown that germs will pass unwetted and unscathed through sulphuric acid unless the most special care is taken to retain them.—See Tyndall's article on Spontaneous Generation in the Popular Science Monthly, Feb., 1878, p. 479.—Trans.]

with a porcelain tube stuffed with bits of broken porcelain and asbestos, and made to pass through a hot oven; this tube was connected at its distal end with a number of Liebig's bulbs filled with sulphuric acid. The putrescible substance (urine, roots of plants, beef extract, etc.), enclosed in a strong glass tube, after being heated for two hours at 200° C. (nearly 400° F.), is placed in the horizontal neck of the flask, where it is again heated by the aid of a spirit lamp and kept boiling for a quarter of an hour. The vapor of the water in which the putrescible body is dissolved, traverses the porcelain tube heated to a white heat in the oven, and finally finds an exit through Liebig's bulbs, which contain the sulphuric acid. When the vapor has been largely expelled, the lamp is removed a little from the flask, in order that air may be slowly aspirated from without; it enters the apparatus, traversing first the sulphuric acid in the bulbs, then the labyrinth of the porcelain fragments and filaments of asbestos contained in the horizontal porcelain tube and heated to redness. At length when the flask is at the temperature of the surrounding air, the neck containing the glass tube with the putrescible infusion inside is plunged into cold water. The stop cock is then closed, the flask righted and plunged into an oil bath heated to 300° F.

At the end of a certain time, however, the liquid became turbid and full of fungi and infusoria. G. Pennetier, Mantegazza, Joly, Musset, Schaafhausen, Ingenhousz, J. Wyman, obtained similar results. These are the conclusions of an able advocate of heterogenesis, an opponent of Pasteur.

A. The air is not the vehicle of germs of microphytes or microzoaires: 1. Because they cannot be shown. 2. Because neither the cotton tampon, the germ collector of our amiable antagonist, nor the aëroscope of Pouchet, nor the glycerized glass plates are able to show them. 3. Because ordinary air may be replaced by artificial air, pure oxygen, or air heated to a white heat.

B. Nor is it the water which carries these vile germs, for we are confident, after experiments often repeated, that water submitted to ebullition no longer contains any low forms of life, since all perish at a boiling temperature. Moreover, for this water which has been boiled, one may substitute water obtained by the direct combination of oxygen and hydrogen, and yet life appears in it as in ordinary water.

C. In fine, the germs of which so much has been said, are not any more likely to find their way into the organic substance under experiment, after the manner of the mice and scorpions of Van Helmont, since we take for granted that this substance has undergone the action of a temperature of from 212° to 350° F.

Always and everywhere (and this in opposition to the localized panspermism of Pasteur), when the conditions of life were brought about, whatever may have been the organic material used, we have seen develop, first, bacteria, monads, or spores of yeast and mucedines; then the monadaria have appeared, taking their origin in the proligerous membrane or pellicle formed by the débris of the bacteria, lastly the ciliated infusoria are seen in their turn, and the life stops there.

"But in order to study life," says Joly, "for Heaven's sake! do not begin by suppressing it or putting it to the torture!"

"You are wrong," says Pasteur. "If you had had care thoroughly to exclude the germs of the air, your flasks would have remained sterile."

"The germs of the air, show them," replies Pouchet.

On this point Pouchet and his school have been beaten.

Pasteur and Tyndall have in fact shown corpuscles of form and aspect like cryptogamic spores, in a beam of luminous rays from the sun or the electric light, made to traverse ordinary air. You see in this sunbeam corpuscles which everybody has observed in rays of sunshine allowed to penetrate a dark room. The same experiment with air filtered through a pledget of gun-cotton no longer gives a luminous track because it finds nothing to illuminate in its passage; the air is optically pure.

Let us now, after the example of Tyndall, take a wooden apartment of any given dimensions,[1] thoroughly air-tight, closed, but containing on its two opposite sides two glass windows, which permit a sunbeam to pass through. Cover its floor with a sticky coating of glycerine. At the beginning, the air within this apartment will appear under the action of the solar rays as luminous as the air outside. But little by little, the room being hermetically closed, the dust will fall to the floor, and will be entangled and retained by the glycerine, so that at the end of several days the air will become optically pure. It will then contain all its principles, save the solid matters held in suspension, which are precipitated. You may even leave this room in communication with the outer atmosphere if you wish, provided that air can only be admitted through a tube bent several times on itself, and terminated by a cotton plug, so as to prevent the introduction of particles of dust. Into this apartment you may then introduce any putrescible infusion whatever, on condition that it has been boiled, and leave it there as long as you wish, and no organism will make its appearance in it. It will remain sterile, although if freely exposed to the surrounding air, in forty-eight hours it would be found swarming with life.

We have here the reproduction on a large scale of Pasteur's experiments.

But these corpuscles—are they really living things?

If you take them and sow them in a suitable nutrient liquid under the microscope, so that you can watch them with the eye, you will see them germinate and multiply. They are then really living beings.[2]

But it is necessary to take a step farther and show that it is indeed to them that is due the fecundity of the infusions. Sown in infusions previously sterilized, these bodies gave to Pasteur abundant crops of microorganisms. A flask containing an organic infusion of some kind is connected by its tapering neck with a metallic tube placed in a small furnace, and heated to redness. In bringing the infusion to ebullition, you drive out the air of the flask, and you kill the germs which may exist there, whether on the walls or in the infusion. Allowing the liquid then to cool, the air enters little by little, becomes calcined in contact with the red hot tube and penetrates the flask. As soon as this is full of air, and cold, the neck is hermetically closed by heating it in a spirit-lamp. Thus prepared the flask remains sterile. There is nevertheless in it, water, air, and a putrescible organic substance.

Why does no living organism develop in it? What is wanting, say the panspermists, is the living dust which one may collect on a pledget of gun-cotton placed in a tube through which is drawn a current of atmos-

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[1] Spontaneous Generation, in Revue Scientifique, no. 51, June 22, 1878, p. 1204.

[2] Duclaux, Article on Fermentation, in Dict. Encyclop. des Science Méd., t. i., 4th Series, p. 560.

pheric air by the aid of an aspirator; germinal corpuscles appearing in the midst of fragments of soot, of charcoal, starch globules, débris of clothing, or vegetable matters. If it be pretended that the cotton tampon plays a part in this phenomena, one may reply that if replaced by previously calcined asbestos, and then employed as a plug, you obtain the same result.

Take another example as a counter test. Pasteur places in a glass flask a very alterable infusion, such as urine or a filtered decoction of yeast, and then in the flame of a spirit-lamp draws out the neck of the flask, twisting it into the form of a letter S. He then raises to a boiling point the liquid inside till the vapor issues by the tapering end, then lets it cool. No organism develops in a flask thus treated, although it is allowed to remain in communication with the outer air. One cannot imagine anything in the latter which does not exist in the air of the flask, except the germs which are no longer present in the interior, having been killed by the hot vapor. When you arrest the ebullition, the exterior air enters with force, but it meets a liquid still very hot, almost boiling, which has killed the living germs. When the liquid is cooler, the entrance of the air becomes sufficiently slow so that it abandons in the moist flexures of the neck and in the sinuosities of the bent tube which it has to traverse, the dusty particles of the atmosphere which are capable of fecundating the infusion. The flask remains unaltered.[1]

But if the open extremity of the neck of the flask be now closed in the spirit-lamp, and the flask so tipped that a drop of the liquid in the flexures be made to fall into the infusion, we see then appear the same organisms as if we had left the flask exposed to the dust of the atmosphere. By this experiment it was shown that it was no particular condition of the air as *such* that provoked the microbiosis.

Nevertheless it might still be objected that the experimenter was operating on boiled liquids. Now these, if abandoned to free air, become with more difficulty the habitat of organisms than liquids that have not been boiled.

It was to meet this objection that Pasteur undertook to experiment with solutions containing no germs, such as blood, urine, the intact juice of grape, etc.

The means taken to obtain these liquids just as they are in the living organisms was as follows: A glass flask is taken, and connected by a rubber tube to a brass beak whose distal extremity is long and slightly tapering. The flask contains a little water, which is made to boil; the extremity of the brass beak is first put in communication with a platinum tube heated to redness. Then, when ebullition has lasted for some time, the flask is allowed to cool, and become partially filled with calcined air. The stop cock in the brass beak is closed before the flask is quite cool, in order that the air imprisoned within may be under a less pressure than the air outside. The tapering extremity of the tube, previously heated in a spirit-lamp, then cooled, is now introduced into a vein, an artery, or into the urethral canal. The stop cock is opened and the liquid aspirated in sufficient quantity into the flask, this being easy of accomplishment owing to

[1] This experiment, similar in character to that of Pouchet before given, was attended with quite different results. In Pouchet's experiments it is probable that spores attached to the fragments of asbestos, etc., must have remained intact despite the high heat, or after passing through the acid in the bulbs, they may still have penetrated the flask uninjured.—Trans.

the relative vacuum within. The stop cock is then closed. The flask now contains a liquid that has not been boiled, which is putrescible, and in contact with pure air. Neither the blood nor the urine undergoes putrefaction. "Spontaneous generation," exclaims Pasteur, "is then nothing but a chimera!" At the same time, if Pasteur says, "the blood of an animal in full health never contains microscopic organisms or other germs,"[1] Beale replies, "there is not a single tissue or fluid of the organism but has them; not even the blood is free from them."[2]

Nevertheless it appears certain that although the blood may constantly contain a greater or less number of these organisms, they do not accumulate there in *quantity*, and one may with certainty affirm, according to experiments, that their presence in appreciable number is incompatible with a state of perfect health.

These experiments of Pasteur did not silence the heterogenists. "How admit the existence of so many germs in the air?" said Pouchet; "I do not see them by the aid of my aëroscope, and if there were in the air enough germs to fecundate all the infusions, and produce all the putrefactions, the air would have the density of iron!"

Pouchet here forgets that a single infusorial animalcule may produce millions in a brief space of time. One yeast-cell will give rise to 16 millions in twenty-four hours, 256 quadrillions in forty-eight hours, if nothing hinders their development. A cercomonade observed by Dallinger and Drysdale, and which reproduces by fissiparism, engenders more than a thousand offspring in an hour, more than a million in two hours, and in three hours more than there are inhabitants on the surface of the globe. Davaine has calculated that one bacterium introduced into the blood may, in seventy-two hours, give origin to more than sixty millions of bacteria.

Moreover, it would not be true to say that there exist germs everywhere in the atmosphere, and that it suffices to place the smallest quantity of air in contact with an infusion, to cause this to be inhabited by microorganisms. Pasteur repaired to the Mer de Glace, near Montauvert, and there opened a certain number of flasks drawn out and sealed by the lamp, and containing organic infusions. Out of twenty flasks, to which the air of the glacier was admitted, only one a little later gave signs of life, while out of the same number of flasks charged with the same infusions, but to which the air of the plains was admitted, eight soon swarmed with living organisms. Carried to the cellars of the Observatory of Paris, these flasks remained sterile; the still air of these cellars protected from the atmosphere outside, and from all life, neither engendered putrefaction nor living organisms of any kind. Pouchet, Joly, and Musset repeated under the same conditions Pasteur's experiments, on the Pyrenees, at the Rencluse, by the foot of the glaciers of Maldetta, and when on their return to Luchon these savants opened their flasks, "*eight out of every eight*" were full of infusoria or of mucedines![3]

Following in the wake of these contradictory experiments, the dispute was carried before a commission of the Academy of Sciences, but the experimenters did not agree, and the heterogenists withdrew, signing a protestation. (Revue des cours scientifiques, No. 31, July 2, 1864, p.

[1] Compt. Rend. Ac. des Sciences, lxxxv., p. 188, July 16, 1877.

[2] Disease Germs, 1870, p., 64. See, on the other hand, Cunningham and Richard Lewis, who affirm the exceptional presence in blood and other healthy fluids of bacteria.

[3] G. Pennetier, loc. cit., p. 262.

431.) Recently Tyndall (Revue Scientifique, No. 31, June 22, 1878, p. 1202), repeated in the Alps the experiments of Pasteur, and arrived at the same conclusions. He filled a number of clean empty flasks with boiled infusions of beef, mutton, turnip, and cucumber, then placed the flasks for five minutes in an oil bath raised to the temperature of 250° F., then closed the necks of the flasks after gently drawing them out in a Bunsen's flame, by which the canal was completely blocked up. He carried sixty such flasks to the Alps carefully packed in saw-dust. He opened his box at the Bel Alp, and counted out fifty-four flasks with their liquids as clear as filtered drinking water. This was at an elevation of about 7,000 feet above the sea. In six flasks the infusion was found muddy. On examination it was noticed that in every one of these six, the fragile end had broken off in the transit from London; air had entered the flasks and the observed muddiness was the result. Examined under the microscope with a magnifying power of a thousand diameters, this liquid was found swarming with organisms.

Had this multitudinous life been spontaneously generated in these six flasks, or was it the progeny of living germinal matter carried into the flasks by the entering air? "If the infusions," says Tyndall, "have a self-generative power, how are the sterility and consequent clearness of the fifty-four uninjured flasks to be accounted for? It may be urged that the assumption of germinal matter is by no means necessary; that the air itself may be the one thing needed to wake up the dormant infusions. We will examine this point.

"Our fifty-four vacuous and pellucid flasks declare against the heterogenist. We expose them to a warm Alpine sun by day, and at night we suspend them in a warm kitchen. Four of them have been accidentally broken; but at the end of a month we find the fifty remaining ones as clear as at the commencement. There is no sign of putrefaction or of life in any of them. We divide these flasks into two groups of twenty-three and twenty-seven respectively (an accident of counting rendered the division uneven). The question now is, Whether the admission of air can liberate any generative energy in the infusions. Our next experiment will answer this question, and something more. We carry the flasks to a hay-loft, and there with a pair of steel pliers snip off the sealed ends of the group of twenty-three. Each snipping off is, of course, followed by an inrush of air. We now carry twenty-seven flasks, our pliers, and a spirit-lamp to a ledge overlooking the Aletsch glacier, about two hundred feet above the hay-loft, from which ledge the mountain falls almost precipitously to the northeast for about a thousand feet. A gentle wind flows towards us from the northeast; that is, across the crests and snow-fields of the Oberland Mountains. We are, therefore, bathed by air which must have been for a good while out of practical contact with either animal or vegetable life. I stand carefully to leeward of the flasks, for no dust or particle from my clothes or body must be blown toward them. An assistant ignites the spirit-lamp, into the flame of which I plunge the pliers, thereby destroying all attached germs or organisms. Then I snip off the sealed end of the flask. Prior to every snipping the same process is gone through, no flask being opened without the previous cleansing of the pliers by flame. In this way we fill our twenty-seven flasks with the clean vivifying mountain-air.

"We place the fifty flasks, with their necks open, over a kitchen stove in a temperature varying from 50° to 90° F., and in three days find

twenty-one out of the twenty-three flasks opened on the hay-loft invaded by organisms, two only of the group remaining free from them. After three weeks' exposure to precisely the same conditions, not one of the twenty-seven flasks opened in free air had given way. No germ from the kitchen air had ascended the narrow necks, the flasks being shaped to produce this result. They are still in the Alps, as clear, I doubt not, and as free from life as they were when sent off from London.

"What is the only logical conclusion from the experiment before us? Twenty-seven putrescible infusions, first in vacuo, and afterward supplied with the most invigorating air, have shown no signs of putrefaction or of life. And as to the others, I almost shrink from asking my opponents whether the hay-loft rendered them spontaneously generative?

"Is not the inference here imperative that it is not the air of the loft—which is connected through a constantly open door with the general atmosphere—but something contained in the air that has produced the effects observed. What is this something? A sunbeam glinting through a chink in the roof or wall and traversing the air of the loft, would show it to be laden with suspended dust particles. Indeed the dust is distinctly visible in the diffused daylight. Can it have been the origin of the observed life? If so, are we not bound by all antecedent experience to regard these fruitful particles as the germs of the life observed?" (Popular Science Monthly, February, 1878, p. 476.)

By these check experiments of so complete a scientist, experiments so strikingly confirmatory of his own results, Pasteur had reason to felicitate himself in his triumph over his opponents, and in the demonstration of his affirmation that the air is indeed charged with germs, but does not everywhere contain them.

Nevertheless like a new Proteus, which is always changing its form and reappearing, *Spontaneous Generation*, not yet quite annihilated by such experiments as those of Pasteur and Tyndall in the Alps, persists in raising its bold front. In 1875, on the occasion of a communication from Albert Bergeron to the Academy of Sciences, presented by Gosselin (Session of Feby. 15–22, 1875 "On the presence and formation of vibriones in the pus of abscesses which have never been in communication with the exterior air"), the spark was given to the powder and the commotion made itself felt in the learned Society of the street Des Saints Pères. In this connection was discussed the subject of *the putrefaction of eggs with shell intact* which had been demonstrated by the experiments of Legros and Onimus, of Donne, of Béchamp, of Colin of Alfort, but which Gayon affirms to be not the result of spontaneous generation, but of germs which penetrate the egg during its formation and its transit in the oviduct, passing even through the membrane or shell when it leaves the oviduct, (the eggs without germs, for some do not contain them, and these do not putrefy according to Pasteur); there came up also for discussion the experiments of Bellamy and LeChartier on the alcoholic fermentation of intact fruits and grapes exposed to the air or plunged in an atmosphere of carbonic acid—a fermentation which Pasteur explains by saying that the cells of the parenchyma of the fruit act like yeast—they partially transform their sugar into alcohol and carbonic acid; there was also reference made at this time to the ammoniacal fermentation of urine in the bladder and even in the kidneys (Gosselin), which Pasteur does not explain, but in regard to which he says: "whenever urea undergoes transformation into carbonate of ammonia, you will find in the liquid (and clinical experience

bears me out), an organized ferment (a torula)." "Then if this view be correct," Colin replies, "we must admit continual fermentations in certain herbivora which always have alkaline urine." (See the Bulletin of the Academy of Medicine, Feby. and March 1875.) But, as Poggiale has remarked, one may always reply to the partisans of spontaneous generation: "the germs of vibriones, of bacteria, of mucedines, of the ferments whose presence you have noted in the organism, have been introduced by the mucous membranes, by the respiration, etc., thereby impregnating your whole body to its deepest recesses." Nevertheless, if there be no ferment without fermentation, it may be said that there are fermentations without ferments; such is the case with the alcoholic fermentation of glucose in the tissue of apples in the process of ripening and in the fungi known as hymenocetes.[1]

Within the last decade, Pasteur has found opponents in Dr. H. Charlton Bastian, Huizingar, J. Wyman, etc.

Bastian takes urine, and after having boiled it, preserves it in presence of calcined air. Nothing is developed in it. This is the experience of Pasteur and his followers. He then saturates it with a solution of potash, boiled and deprived of germs, and exposes the whole to a temperature of 120° Fahr. The urine thus treated becomes fecundated in several hours. But as in this experiment if you substitute for the solution of potash, a fragment of solid potassa previously heated to redness, the urine remains sterile, Pasteur deems himself warranted in affirming that it is the solution of potassa employed which has brought the germs. Bastian indeed says that he carried it to ebullition; but sometimes a temperature of 212° F. is insufficient to kill the germs. Milk needs to be heated to 250° F. in order to become sterile, and it is the same with alkaline yeast water. There is, then, nothing astonishing in the fact that there were germs in Bastian's solution of potash. In fact, is it not prepared with ordinary water, and has not Burdon Sanderson shown that no water is exempt from germs?

Tyndall takes two boiled liquids, sterilized and long remaining limpid; one of these liquids is a mineral solution fit to support the life of bacteria, the other is an infusion of turnip or some other animal or vegetable substance. He takes one drop of a putrid liquid (juice of meat undergoing putrefaction) which is swarming with bacteria, and inoculates the two sterilized solutions. At the end of twenty-four hours they are teeming with micro-organisms. Then he varies the experiment. He opens a room which has been kept hermetically closed, and where have remained for months two flasks containing similar sterilized liquids, and he casts into these flasks a pinch of dust from his laboratory. The result this time is less rapid. At the end of three days, however, the infusion of turnip is full of bacteria, but the mineral solution remains clear. What conclusion is to be drawn from these experiments? It would seem that the two liquids are suitable to nourish bacteria fully formed, while the organic liquid alone is capable of developing into active bacteria the organic and germinal dust of the atmosphere.

Now certain scientists have come to a different conclusion. As the mineral solution nourishes bacteria when once formed, and as, on the other hand, no bacteria develop there after the solution has been long exposed

[1] Le Chartier and Bellamy, Compt. Rend. Ac. des Sc., Paris, 1872, t. lxxx., i., p. 1204. Also Muntz in ibid., 1875, t. lxxx., p. 180.

to the air, they have said there are neither bacteria nor germs in the atmosphere. Hence, if only the organic infusion after exposure to the air develops bacteria, these must arise spontaneously by a special chemical process. (C. Bastian, Proceedings of the Royal Society, vol. xxi., p. 130.)

But Tyndall replies: "Why, then, when the sterilized organic infusion is in contact with air optically pure, does not this biogenesis take place?"

It is a little singular that the last experiments of Claude Bernard were not in favor of panspermism.[1] He formulates the following, among other conclusions deducible from his experiments: "alcohol is generated under the influence of a *soluble ferment*, apart from the agency of any micro-organism, in fruits ripening or rotting; there is simply decomposition of the fruit, and not biotic synthesis of yeast or of vegetation. The soluble ferment is found in the juice extracted from the maturing fruit. The alcohol continues to form there and to increase." Pasteur, attacked in his stronghold, was much disturbed by the publication of this posthumous note of Claude Bernard, and on this subject there arose a spirited discussion between him and Berthelot at the Academy of Sciences.

From a communication of D. Monnier and Carl Vogt to the Academy of Sciences (Session of January 2, 1882), we cite the following startling conclusions which, if they should be confirmed, would go far to relegate the mysteries of living creatures to the ordinary physico-chemical laws of the universe:

"Certain figured elements presenting all the characteristics of form pertaining to the organic elements, such as simple cells and porous ducts, tubes with walls and septa, and heterogeneous granular contents, etc., may be produced artificially in an appropriate liquid, by the concourse of two salts, forming, by double decomposition, two, it may be only one insoluble salt. One of these salts must be in solution in the liquid, while the other ought to be in a solid form.

The forms of organic elements (cells, tubes, etc.), being a production from liquids absolutely inorganic (such as silicate of soda), as well as from liquids of organic or semi-organic nature (of the latter we have an example in sucrate of lime), it can no longer be a question of distinctive forms characterizing organic bodies on the one hand and inorganic bodies on the other.

The formation of pseudo-organic figured elements depends on the nature, on the viscous constitution, on the concentration of the liquids in which they are produced. Certain viscous liquids (solution of gum arabic, of chloride of zinc), give none.

The form of the pseudo-organic products is as constant as the crystallization of minerals. It depends principally on the acid which enters into the composition of the solid salt.

The artificial pseudo-organic elements are surrounded by veritable membranes, which are eminently *dialysant*, allowing only liquids to pass through. They display heterogeneous contents, and produce in their interior granulations arranged in a determinate order. They are then, with respect to their constitution, as to their form, absolutely like the figured elements of which organisms are made up. If these results are confirmed, we shall be able to say that certain figured elements—primary organisms—may come into being spontaneously by virtue of simple chemical processes,

[1] Rev. Scien. No. 3, July 20, 1878, and Discussion between Berthelot and Pasteur, Compt. Rend. Acad. des Soc., Nov. and Dec., 1877, and Jan., 1879.

unknown, indeed, but constantly operative in causing matter to pass from a dead to a living state."

Science has not, then, said its last word on this vexed problem of the ages; for, as Charles Robin observes, before seeking to comprehend the formation of any animal whatever, of however inferior a grade, you must first settle the question as to the genesis of a single anatomical element. Now it is manifest that certain elements, such as the vitelline nucleus in the centre of the ovule after fecundation, vegetal cells in the embryonic sac, spores in the thecæ of fungi, are formed, to all appearances, spontaneously in the midst of organized, though, it is true, living matter.

"But," one may say, "how happens it that these microscopic beings, which surround us and besiege us in all directions, which penetrate our very blood and our humors through our bronchi and our blood-vessels—how happens it that these pestilent parasites, which have been found even in the pus of an abscess in the brain, do not inflict upon us still more harm? How do we resist successfully in the struggle for existence with these invisible enemies?"

The reason is just this, that these little beings cannot develop, live and multiply except in certain media; and an organism already in a pathological state is (generally) a necessary condition. If this be the case, is it not a legitimate supposition that they are only an epiphenomenon in diseases and not a cause? This is in fact the opinion of some savants. Nevertheless as far as chicken cholera and anthrax are concerned the question is settled. Pasteur has found, cultivated, and inoculated the microbe, and always with the same success. The same may be said of other parasitic diseases in animals and in vegetables; in reference to the tubercle bacillus, the causal relation also seems to have been established by Koch's experiments. We shall return to this subject further on. With reference to spontaneous generation, it would seem that in the actual condition of science, this has not been proved, and the most serious arguments which one can oppose to the heterogenists are perhaps, as Prof. Girard has remarked,[1] drawn from morphological considerations. It is in fact impossible for any one who has studied with care the organization of the infusoria and even of the Protista, to admit that beings so complex can ever have been formed by spontaneous generation. The imperfection of micrographical researches alone has allowed the admission of the genesis of such beings as the Paramecia or the Mucedines. Even in the case of the Bacteria, the hypothesis of heterogenesis seems to be negatived by this observation, that these beings present complicated metamorphoses. This is what has been proved by the researches of Ray Lankester and Girard on the colored vibriones. An evolution, that is to say, a series of metamorphoses, supposes a special state of the germ, the result of heredity, and consequently proves a generation dependent on other antecedent organisms.

Reasoning, however, leads us to conclude that the first living beings must have been formed independently of any pre-existing organism.

When the terrestrial globe first began to cool, before the appearance of green vegetables, the atmosphere was very rich in carbonic acid, water, oxygen and hydrogen, and in nitrogen; under the influence of the high temperature and powerful sources of electricity, numerous combinations must have been produced among these elements; first carburets of hydrogen, then azotized combinations more or less similar to the albuminoid matters with which we are acquainted.

[1] General Principles of Biology, in Huxley "On Invertebrate Animals," p. viii.

Chevreul, Berthelot, and Schutzenberger have been able from the very elements to effect the synthesis of vegetal essences, of alcohols, of fatty bodies and of urea. Berthelot has experimentally proved the transformation of ternary into albuminoid bodies under the influence of electricity. Is not every one familiar with Traube's artificial cell of tannate of gelatine which assimilates, disassimilates, and develops by intussusception? Is there an enormous difference between these proteinoid substances and the most elementary living molecules or even the most primitive living being? Among the innumerable combinations of this sort which nature undoubtedly produced during the series of ages when the earth was gradually cooling, many must have persisted during the period when already, the water being condensed into a fluid, there were seas on the surface of the globe. The congeries of these substances or of such as may act chemically on each other, and regenerate themselves at the expense of proteinous materials which surround them, or even at the expense of more elementary compounds (for Pasteur, Raulin, and Gayon have shown that the most simple organisms may live in certain media exclusively mineral), must have constituted the first living beings, beings of extreme homogeneity and scarcely comparable with the organisms which we term Monera.[1]

Without doubt the first forms of life must have been much more simple even than a monad, an amœba, a gregarina, or a bacterium; but these lowly forms may at the same time give us an idea of the passage from the mineral to the living world. What are they, if not simple masses of albuminoid substance, undifferentiated globules of protoplasm, without special organization and without constant form? What is this protoplasm, this "physical basis of life," as Huxley calls it, this substratum of every cell, but an aggregation of albuminoid molecules, of high chemical complexity and unstable equilibrium.

The moner is only a minute speck of granular protoplasm, without nucleus or enveloping membrane such as we see in the perfect cell. It assimilates, disassimilates, reacts, and contracts under the influence of excitants, and reproduces by segmentation; in a word it *lives*. Now every living being, plant or animal, is nothing but an agglomeration of cells more or less differentiated (elementary organisms), grouped in tissues, in organs, in systems, and adapted to different functions, and it is not merely a small number of exceptional organisms which present themselves under this simplified form of a globule of protoplasm—all living beings and man himself are in just this very state in the egg before fecundation. And in this protoplasm there is nothing but a complex mixture of immediate principles, quaternary bodies, ternary and earthy compounds, nothing but H. O. C. N. and a few other simple accessory elements such as S. P. Na. K. Ca. Mg. Fe.

Such are the elements which by successive and very unstable combinations, come to form the substratum of life. There is no element in the living body which is not found in, and which does not come from inorganic nature, whither it returns at death, but only to enter upon a new life. And what is chemical synthesis but a first trace of organization? Life itself is only a series of chemical combinations and decompositions which produce heat, force and movement; only a series of organic formations, evolutions and destructions. It has its explanation in the physico-chemi-

[1] "The simplest of all observed organisms; *organisms without organs;* which are nothing but simple little lumps of an albuminous condition of carbon," Haeckel, Natural History of Creation, vol. i., p. 343 (Am. Ed.).—Tr.

cal constitution of protoplasm—living matter capable of regenerating itself at the expense of the environment in which it is placed, in strict correlation with the ejection into that environment of excreta produced by the dynamic manifestations which it engenders. Living matter may then be roughly compared to an electric pile whose elements are capable of regenerating themselves indefinitely.

Moreover, unless we are ready to admit Creationism, unless we are ready to believe that something can come out of nothing, an absolute nothing being inconceivable, we must admit—and reasoning forces this conclusion upon us, that the first living beings came out of the womb of inorganic nature by natural forces alone. Like crystals set free in the midst of a solution, they were developed in a liquid where were already formed ternary and quaternary compounds, and under the sole influence of reciprocal attractions.[1]

Rôle of the Microscopic Organisms (Microbes or Bacteria) in the Production of Diseases.

The first question which presents itself is is: Is the specific material of infection—the materies morbi of contagious diseases, constituted by living beings? If the air is the vehicle of infectious agents, it will not do, says Nägeli, to believe that the contagious principle is gaseous. In fact gaseous matter would be rapidly diffused through the whole atmosphere; in such a state of diffusion, it would soon fail to have any action whatever.

On the other hand, if the contagium were of such a character it would diffuse itself through the air of an entire quarter, of an entire city, and even of an entire country, in such a manner that all the inhabitants of those places would have to breathe equal quantities of the gaseous substance. In this event, either nobody would become sick or everybody would be sick. But the truth is that often the contagion is limited to a house or to a quarter of a city, nor are even all those who are predisposed to take the disease attacked by it; those only take it who have absorbed the infectious matter.

What then is this material of contagion? There is every probability that it is of a nature resembling the minute micro-organisms and their spores, which as we have before shown, flit about unequally diffused in the air in the state of corpuscles, and which cultivation and experimental inoculation have proved to be organized.

In fact, only living organisms are capable of producing the effects which one observes in infectious diseases. The absorption of an amorphous

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[1] [This view, held as a matter of speculative belief by Huxley, Spencer, Tyndall, Haeckel, De Lanessan and hosts of eminent scientists, is earnestly repudiated by Pasteur, Lionel Beale, etc.

On the subject of "Spontaneous Generation," consult the following writings: Pouchet, New Researches on Spontaneous Generation and Vital Resistance, Paris, 1864, and other scientific papers by M. Pouchet; Joly, Critical Examination of the Memoir of M. Pasteur, relative to Spontaneous Generation, Toulouse, 1864, etc.; Mantegazza, On Spontaneous Generation, Milan, 1864; Pasteur, Conférences in Sorbonne on Spontaneous Generation, Rev. de Cours. Soc., April 23, 1884, p. 205; Tyndall, published works and lectures in Popular Science Monthly: Huxley, Lay Sermons and Reviews, 1871, article, Spontaneous Generation; Spencer, Principles of Biology, Appendix in reply to the North American Review; H. Charlton Bastian, Beginnings of Life, 2 vols., New York, 1872; Lionel Beale, Protoplasm; or, Life, Matter and Life, London, 1871; Claude Bernard, Phénomènes de la Vie., p. 223, etc.]

poison either immediately causes sickness and death by its great quantity or remains inactive by its small quantity. It is not thus with infectious agents. An infinitesimal quantity, one bacterium, may be introduced into the organism; it does not give rise to immediate morbid symptoms, but possesses the faculty of multiplying itself in the system, to cause at a given moment troubles more or less grave. We have here the period of incubation.

It is true that in admitting the "theory of contact" as one origin of fermentation, the above phenomenon may be explained quite differently, and that one may be warranted in maintaining that the absorption of an amorphous and non-living toxic matter may provoke the morbid fermentation and the clinical syndrome by which it reveals itself.

"It is impossible," says Prof. Cohn, of Breslau, "for an azotized substance to enter into putrefaction (putrid fermentation) if you destroy its bacteria, and prevent new organisms of this kind from entering." This goes to confirm the views of Pasteur.

Nevertheless, a pupil of Pasteur, M. Gayon, has demonstrated the formation in eggs of the products of putrefaction of albuminoid matters (tyrosine, and leucine), although it was not possible to find in these eggs the least trace of inferior organisms. Now, according to Schutzenberger, the presence of tyrosine and leucine is the palpable proof of putrid fermentation, by this term being understood the decomposition of albuminoid matters, or the transformations which are produced in bodies after death.

What Schutzenberger says with reference to putrid fermentation confirms, then, what we have said before, to wit, that the fermentations called direct, although habitually and incontestably produced under the influence of microbes, have nevertheless nothing special and absolute about them, since they may equally be provoked by quite different chemical agents. If you fix well in the mind these principles, you will readily understand that Pasteur goes too far when he assigns to each fermentation a special organism-ferment. Have we not seen that the acids act like the salivary diastase or the pancreatic juice? Is it not known that the alcoholic fermentation may be caused by different species of saccharomycetes, and even by certain moulds, *Mucor-racemosus*, *Mucor mucedo*, *Penicillium-glaucum*, etc., and by the cells of divers organs of vegetables?

Pasteur, however, in his communications made only a short time ago to the Academy of Sciences,[1] has defended this hypothesis (now becoming so widely entertained) that every virulent disease has its particular species of bacterium, and he particularizes the microbe of charbon discovered by Davaine, and the microbe of *fowl cholera*. But epidemic diseases have a limited duration in the history of humanity; their form changes sometimes from one season to another, from one epidemic to another. It would be necessary, then, to admit a disappearance of certain microphytes, and the appearance of other species and the frequently rapid transformation of these. Assuredly species become modified, but the time taken in the transmutation is infinitely longer than that which appertains to the transformation of a disease. It is true that the experiments of Chauveau, of Pasteur and of Koch on the attenuation of virus may give some light on this subject. Nägeli himself admits alternating generations, morphologically and physiologically distinct, which produce at one state of metamorphosis the lactic fermentation, at another state, the butyric fermenta-

[1] Sessions of July and October, 1880.

tion, under one form the putrid fermentation, under another the development, sometimes of diphtheria, sometimes of recurrent fever, sometimes of cholera, etc.

However this may be, animal and vegetable matters which have ceased to live, disappear as a consequence of transformations over which preside direct combustion by the oxygen of the air and putrefaction. It is especially to this latter agency that is due the destruction of organic substances.

Now the inception of putrefaction is coincident with the appearance and vital operations of vibriones. In this phenomenon there appears on the surface of the matter undergoing putrescence, whether solid or liquid, a gelatinous scum which swarms with bacteria; the *bacterium termo*,—which, note in passing, is destroyed when introduced into the living organism,—the *monas crepusculum*, the *spirillum*, certain long rod-like forms sometimes soldered together and articulated, and certain infusoria such as the *kolpodes*. When all the free oxygen is absorbed, these infusoria perish, unless indeed they can continue to live on the surface of the putrescible material where there is still oxygen, thus intercepting this gas in its passage to the interior of the mass. Then vibriones manifest themselves, similar in aspect to those of the butyric fermentation, and which are very minute and animated with lively movements, species which have no need of oxygen to live, and are even killed by its presence; thus the putrid fermentation declares itself. These vibriones, like the ferments, seize upon the combined oxygen and transform the azotized matters into products simpler, though still complex.[1] Apart from contact with the air, these products may be preserved unaltered, but with free access of air there appear anew bacterium termo and other bacteria and infusoria which attack these organic substances and end in destroying them and restoring their elements to the atmosphere and the mineral world.[2]

These profound transformations accomplished by the bacteria in dead organic materials,—may they under certain conditions develop in organisms which are still subject to the dominion of life? What are the disorders which result from their presence and their multiplication? By what means may one defend himself from their attacks? It will be seen presently what interest attends the response to these questions.

The Pathogenetic Schizomycetes.

According to a view which gains every day new partisans, a contagious disease may be defined as a conflict between the subject who is smitten and a particular organism which multiplies at his expense, appropriates his air and his water, disintegrates his tissues, or poisons him by the decompositions which accompany its development.[3]

These organisms, as we have said, do not come by spontaneous generation from the midst of matters in decomposition. They arise from germs which flit about in the atmosphere, which attach themselves to all objects, and which float invisible in ordinary waters. The waters of springs at the moment when they issue from the entrails of the earth, the tissues and the internal liquids of vegetables and animals, do not contain them. You may in fact, says Chamberland, inoculate sterilized organic infusions such as meat broth with aqueous humor, fresh urine, spring water, juices of

[1] See Davaine, Dict. Encyclop. des Soc. Méd., vol. viii., p. 29.
[2] See Duclaux, Ferments et Maladies, Paris, 1882.
[3] See Duclaux, *loc. cit.*

fruits, cerebral, splenic, hepatic matter, muscular juice of animals, and you shall see no change whatever produced.[1] You may even collect in flasks heated in a gas furnace to the temperature of 307° to 390° F., and protected from germs of the air by a cotton plug, the lymph, blood, milk, and other putrescible liquids of the economy, and you shall see these liquids preserve themselves indefinitely without showing any trace of microscopic organisms.

These germs, then, surround us on all sides. How then at each instant is it that we who breathe them, we who swallow them, do not succumb under their pernicious influence? "Ah," says one, "the air which you breathe, the water which you drink,—are they not filtered by the organs of digestion and of respiration, as is the air which passes through the tampon of gun cotton in the sterilized flask?" "But even if you inject them into the blood, no injurious results necessarily follow, and in fact all the schizomycetes have been thus introduced into the organism with entire impunity.[2] "Two events may happen," reply the advocates of the germ theory; "either these germs will not find the conditions proper for their life and their reproduction, and hence will perish, or they will find a favorable environment and they will then multiply with rapidity and invade a part or the whole of the body of the animal, causing there disease and often even death: whether because these bacteria have given rise by their disassimilations to veritable poisons, like ptomaines, as Nägeli supposes, or because they have taken from the cells of the organism the elements necessary for their life."

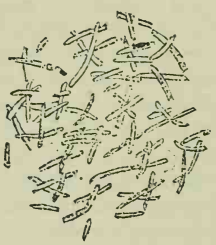

FIG. 14.—Bacillus Anthracis. From the blood of a sheep that died of splenic fever. × 600.

It is in fact true that if you introduce a few drops of a spoiled infusion *i. e.* an infusion full of microbes, under the skin of different animals, sheep, hares, guinea-pigs, fowls, etc., you often observe in some of them symptoms more or less grave. Sometimes these morbid manifestations take the form of abscesses, lymphangites, œdemas, which extend over a larger surface, and the animal is sick for several days, but recovers; sometimes also it succumbs to a veritable infection comparable in certain respects to the purulent infection (experimental septicæmia); at other times, however, although there is produced no local disorder, the animal succumbs and its blood and tissues are found full of microbes. It seems certain that it is these microscopic beings, introduced into the subcutaneous cellular tissue, which are the cause of the disease and of death, for if you heat these liquids so as to kill all the organisms before you inoculate with them, you observe nothing but an insignificant local disorder.

[1] Chamberland, Conference at the Annual Congress of the Scientific Association of France. April, 1882. Rev. Scient., May, 1882. Tribune médicale, Nos. 714, 715, 718, 719, 720, May and June, 1882.

[2] *Vide* Richard Lewis, The Microphytes of the Blood and their Relations with Diseases, Rev. Int. des Soc., No. 6, June 15, 1880.

This variable resistance which is noticed in the different animals, may be due to the nature of the cells and of the intercellular medium. It may be that in this case there is established a struggle for existence between the cells of the living body and the bacteria, in which, as always, the weakest succumb.

Have we not here the explanation of the accidents which supervene so frequently as a sequel of wounds or surgical operations? Always there is a little blood and lymph which exude from the wound. These putrescible liquids in contact with the air and the dressings become filled with vibriones, like Pasteur's infusions; many of the bacteria are inoffensive because they cannot develop within the organism of the subject; but if certain of them should happen to be, what Pasteur calls *anaërobiotic* and find in the interior of the economy a favorable environment for their evolution, they will pullulate and produce disorders more or less grave.

Let us come now to the facts.

"Charbon[1] is the disease of the bacterium, as the itch is the disease of the acarus," says Pasteur.

FIG. 15.—Bacillus Anthracis. From the blood of a guinea-pig. × 650.

In many countries, as in France and in Eastern Europe, it too often happens that flocks of sheep are, without apparent cause, smitten with a most fatal disease.

Out of a flock of two hundred sheep, for instance, there will die rapidly and as if struck by lightning, two to-day, four to-morrow, eight next day, ten, twenty, and so on, the following days. At times in Russia this epidemic has been so disastrous as to obtain the appellation of "Siberian plague." In such conditions, the greater part of the herds invaded would have quickly succumbed to the disease, if the proprietors had not found a long time ago that in changing the flocks from place to place, in removing them to other pasture grounds, and especially in isolating the healthy animals from those that are sick, the epidemic may be cut short.

The necropsy of the animal killed by the disease shows the blood to be black and glutinous, and discloses an enormous and softened spleen, which has given to this affection the name of *sang de rate* (spleen-blood).

Formerly in order to explain the appearance of this plague, pathologists alleged such common causes as the nature of the soil, of

[1] Charbon; Sang de rate; Milzbrand of the Germans; Anthrax, or Splenic Fever of the English; Malignant pustule, etc.

the waters, of the fodder, excessive heat, wet seasons, etc. It was known, however, that this disease was inoculable, and that it might be communicated to man either through contact by dressing the skins of animals that had fallen victims to the disease (tanners being frequently subject to it), or by the sting of a fly, [the latter being frequently the bearer of the contagion]. This was a first fact towards the etiological solution of the question; but it had long been known that canine rabies, yellow fever, small pox, malignant typhus, etc., were equally transmissible, and nevertheless we are scarcely even now more advanced as to a knowledge of their causation. It is no longer so in the case of malignant charbon, thanks to the labors of Pasteur.

Examine the blood of a sheep that has died of charbon. You find the globules deformed, as if running together (blood sticky and glutinous), and between these heaps of corpuscles certain cylindrical rods, straight and rigid, often composed of several segments, and of the length of 10 μ. to 12 μ., shown for the first time by Davaine in 1850; these are the bacteridia of Davaine; the name given by Cohn is *bacillus anthracis*. But it was not till 1860, and from 1860 to 1864, after Pasteur had proved that the butyric fermentation was due to certain vibriones having the greatest resemblance to the filiform bodies found in the blood of animals dead from charbon, that Davaine suspected that these filaments (bacteridia) might be the cause of the disease.

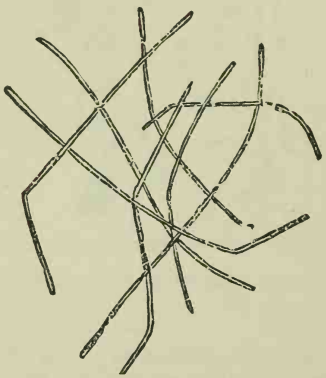

Fig. 16.—Bacillus anthracis. From the spleen of a mouse after three hours' culture in a drop of aqueous humor. (Koch.) × 650.

He then inoculated animals with the infected blood, and noticed that even in very minute quantities this blood was capable of causing death, and always he found innumerable bacteridia in the blood. Thereupon reasoning from the analogy of what takes place in the case of fermentation of dead matter, he did not hesitate to conclude that the disease must be attributed to these little organisms. But meanwhile Leplat and Jaillard, then Sanson, and Bouley, pretended that the inoculation of the blood taken from animals sick with anthrax might cause the death of the animal inoculated, without it being possible to observe any bacteridia in the blood, And on the other hand, Coze and Feltz, finding bacteria everywhere, were led to suppose that these little beings were only epiphenomena, and not the cause of the infectious diseases.

More recently, in 1876, Paul Bert having observed that oxygen subjected to a certain pressure kills all living beings, and in particular the

bacteria, had subjected anthrax-blood to a pressure of ten atmospheres of oxygen, and in inoculating this blood, deprived as he believed of all bacteria, he had induced the disease fatally in guinea-pigs, in the same conditions as with blood which had not been submitted to this pressure; and what seemed further to prove that the bacilli were dead, killed by the compressed oxygen, was that the animals inoculated with blood thus treated, presented no bacilli. Paul Bert arrived then necessarily at the conclusion that the anthrax blood, when deprived of bacilli, might transmit the disease, and he compared the morbific principle of this blood to the soluble ferments such as diastase, pepsin, the venom of scorpions, vaccine virus, the virus of glanders, etc., which are also not arrested in their functions by compressed oxygen.

Here Pasteur, attacked in his main thesis, comes in with his explanation. In collaboration with Joubert, he endeavored to separate the bacteridia from everything foreign to them in the blood, in order to show that the charbon is the result of the microbes exclusively. For this purpose he made successive cultures of a drop of charbon-blood sown in a sterilized flask of yeast infusion neutralized by potassa. Now he had calculated that at the tenth culture the drop of blood would become diluted in a volume of liquid greater than that of the whole earth. Nevertheless a single drop of the product of the tenth, twentieth, even fiftieth culture, injected under the skin of a sheep, gave it splenic fever just like a drop of the original infected blood. The charbon seemed, then, to be unmistakably the disease of the bacillus.

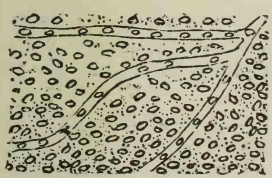

FIG. 17.—Bacillus anthracis, spores and plants.

FIG. 18.—Germination of the spores of Bacillus anthracis.

In these cultures Pasteur and Joubert noticed that the bacilli underwent modification; they were no longer in the form of rods 12 μ. in length, as in the original anthrax blood; on the contrary, they became longer, curled in places, and filled with refractive nuclei, which at a given moment became detached and floated in the liquid. These nuclei were the germs, the spores, or the seeds of the bacteridia, which Dr. Koch, of Wollstein, had pointed out in 1876,[1] which J. Cossart Ewart[2] had found, and which, placed in broth or aqueous humor, reproduced the bacteria filaments. The charbon blood taken from the body of an animal and exposed to the air, behaves exactly like the artificial culture liquid just described.

The bacteridia exist, then, in two states: under the form of rods and under the form of spores or germs. Now to these two states there belong quite different properties, say Pasteur's followers. The bacteridium is killed by a temperature of 140° F.; it succumbs under the action of the vacuum, of compressed air, of desiccation, of carbonic acid, and of alcohol. The

[1] Cohn's Berträge, Band 1, Heft 3.
[2] Quarterly Jour. of Microscopical Science, April, 1878, p. 161.

germs, on the contrary, resist all these agencies, and a temperature of 194° to 203° F. They float about in the air, withstand desiccation for years, and if they light upon a favorable medium, may develop.

This distinction explained a fact which thus far had remained in great obscurity. Davaine had shown that the contagious property of anthrax blood disappeared at a certain time, and did not resist putrefaction. How, then, could the facts be interpreted of the transportation of the disease to great distances, its reappearance and apparently spontaneous outbreaks at the commencement of every epizoötic epidemic?

This mystery was due to the fact that it was not known that the bacteria have two modes of existence, one of active life, under the form of rods, another of latent life, as spores or corpuscle germs, whose activity may remain dormant for a long time, but which may be awakened with all its pestilential power, when more favorable external circumstances permit the development of new animated filaments.

Pasteur then convinced Paul Bert that, although he had succeeded in killing the living bacilli, he had not destroyed their spores. Thus on adding to neutralized urine a little of a dry alcoholic precipitate of infected charbon blood, he proved that the liquid not only possessed virulent properties, but still presented numerous bacteria, and the conclusion was that these bacteria came from the spores contained in the alcoholic anthracoid precipitate, and which afterwards developed into fatal microbes. It was probably the same, so Pasteur argued, with the blood subjected to compressed oxygen.

It will be seen, then, that in admitting without reserve the views of Pasteur, we have to infer the existence of the germs of the bacillus, which have not as yet, however, been directly *demonstrated* in these cases, and, moreover, we have to admit that they resist influences which are destructive to all other forms of life.

It is true that Tyndall pretends that not even eight hours' boiling always kills the bacteria, and he refers in this connection to the communication of Pouchet to the Academy of Sciences in 1866, concerning grains of cereals brought from Brazil, which germinated after being boiled four hours.

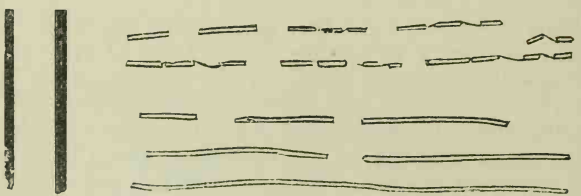

Fig. 19.—Rods and filaments of the bacillus anthracis undergoing segmentation. (Ewart.)

Cossart Ewart, however, who noted that these spores did not always survive ebullition and compressed oxygen, contradicts the assertion of Pasteur and Koch. Lebedeff, of St. Petersburg, has also noticed that septic liquids and the inferior organisms lose their virulence when heated in the stove for forty-eight hours up to 104° F. (Biological Society, March 17, 1882.)

Nevertheless, Paul Bert insists that he has kept anthracoid blood in dilute alcohol for eighteen months, and that this blood has preserved all its virulence even after having been subjected to the action of compressed oxygen. In these cases the bacteria were killed, but the spores survived.

It seems, however, difficult to grant that these germs can survive the following experiment of Paul Bert: To some anthracoid blood full of bacteria he adds slowly four times its volume of absolute alcohol, then he filters the mixture and desiccates the coagulum in a vacuum, the vessel having previously been sterilized by alcohol. This coagulum, injected under the skin of a guinea-pig kills it speedily, and the blood of the latter gives the disease to another guinea-pig and to a dog. Nevertheless the blood of all three animals was found destitute of bacteria. (Compt. Rend. Soc. de Biologie, January, 1877.)

Whether one admits the existence of these spores or not,[1] Pasteur has, nevertheless, arrived at remarkable results. With Chamberland and Roux he performed an interesting experiment. Taking his stand on the principle that the bacteridia which are found in so great quantity in the blood of anthracoid animals, the victims of a spontaneous outbreak of the disease, are not generated *de novo* in the bodies of these animals, he fed some sheep on a farm at Chartres with some grass on which had been scattered the germs of the infecting bacteridia. At the end of from four to nine days a certain number of the animals succumbed, and at the necropsy the same lesions were found as in the case of the victims of the spontaneous outbreak. It was then evident that the ingestion of charbon spores had given the sheep the disease.

FIG. 20.—Germination of the spores of bacillus anthracis. (Koch.) × 650.

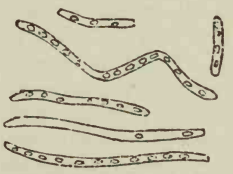

FIG. 21.—Spore formations in the bacillus anthracis. (Koch.) × 650.

Having remarked at the necropsies made by certain veterinary surgeons, that the glands which surround the throat were swollen as if the inoculation had been made in the vicinity, Pasteur supposed that there might have existed little cracks or excoriations on the surface of the mucous membrane of the mouth, and these were the portals of entrance of the germs. To test this theory, he made the sheep eat grass containing barley or wheat beards and thistle barbs, in order to produce artificial excoriations, by which the germs might enter. The mortality augmented in consequence, showing that the inoculation took place by the primæ viæ; but it was evident that the germs might also have pene-

[1] But the existence of these spores has been proved beyond the possibility of a reasonable doubt. Koch's studies on the anthrax bacillus deserve mention here. Davaine and Rayer had observed the rod-like forms in the victims of splenic fever, and Davaine had suggested that they might have a causal relation. Koch's attention was directed to the subject; he investigated the habits of these organisms, and cultivated them in the aqueous humor of an ox's eye. He observed these rods under the microscope (a drop of the aqueous humor being employed) to lengthen out and filaments to form, some straight, some coiled into graceful figures. By and by he saw appear in them little dots which developed into spores. He inoculated both spores and rods in guinea-pigs and rabbits, communicating the disease in a virulent form. Even when the dried blood containing the spores was reduced to dust and kept a long time (four years or more), animals inoculated with this dust speedily died of splenic fever. To Koch, then, belongs the principal merit of having proved the existence and propagation of this disease by minute spores.—Trans.

trated by other parts of the digestive canal, being, for instance, absorbed with the food without loss of their virulence.

There remained then only one thing to do, in order to explain the spontaneous appearance of the disease where the contagion could not be traced, viz.: to find the anthrax germs on the fields where the first outbreak of the disease occurred. Here the experimentation was difficult. It was not possible, in fact, to discover these germs by the microscope or by the methods of culture, for many other microbes, quite inoffensive, resemble the specific anthrax microbe. There remained then but one way out of the difficulty: to lixiviate the soil in order to obtain the fine parts in which the germs would be likely to be found, and to inoculate sheep with the results of this lixiviation in order, if possible, to render them anthracoid. But how would it be possible to do this in the case of fields covering many acres? It was then that Pasteur, Chamberland, and Roux conceived the idea of hunting for the germs in the neighborhood of the pits where had been buried animals that had died of the pest; soil from these regions was taken, was washed, and the products inoculated in guinea-pigs, but these died of infectious diseases which were not charbon. How then would it be possible to isolate the anthrax bacterium from other germs which the soil might contain?

These experimenters then took advantage of the property which the germs of the anthrax bacilli possess of resisting a temperature of from 190° to 205° F., and they heated their lixiviated deposits up to these temperatures. This time it seemed as if all the foreign germs had disappeared, for the animals succumbed to the malignant charbon, and the blood of these animals reproduced the anthrax. At a certain distance from the fossæ the earth ceased to contain these micro-organisms.

The presence of these germs, say Pasteur and his collaborators, is easily explained. When an animal succumbs, he is generally stripped of his skin before being buried, so that the blood is effused in contact with the air, and the bacillus is in a medium favorable for producing germs. But even when the animal has not been skinned before being buried, germs are still found around the cadaver and at the surface of the fossa. One difficulty here presents itself. It is well known that putrefaction destroys the virulence of anthracoid blood. Whence then come the germs in cases where the animals are buried without being dismembered, or even skinned? The explanation given is this: It is true that the bacteridium perishes in the interior of the body of an animal, without producing germs; but it does not perish till after several days. Before its death the putrefaction of the cadaver has given rise to a disengagement of gases, and to an issue of liquids charged with bacteria still living, through the rents which are produced in the skin. Thereupon these microbes in contact with the air give origin to spores.

As for the mechanism by which the germs reach the surface of the pits which contain the buried animals, nothing can be more curious.

Darwin, in discussing the formation of vegetable mould through the action of worms, etc.,[1] has shown what active part is played by simple lumbrics in the formation and changes of the earth's surface. It suffices to mention that more than 10,000 kilogrammes per acre of earth pass through their bodies every year. Now worms of this character, whose little mounds, composed largely of their excrements, may be seen on the

[1] See his treatise on Earth Worms.

surface and around the pits, are nourished by the rank soil in the vicinity of the cadavers, from which, in their return to the surface, they are sure to bring back the germs of anthrax in their digestive canal, and which they then void. Now these germs do not lose their virulence in passing through the intestinal canal of the lumbric, any more than through that of the sheep. It is easy, then, to explain how the spores of the bacillus may find their way to the surface of the ground, a result which is also attained when for any cause the soil is dug up.

But it is not to be supposed that always and necessarily there are anthracoid germs at the surface of the pits where have been buried animals that have died of anthrax. In order that the bacillus may give origin to spores, a certain temperature is necessary. Below 53° F. germs do not form. In the case of anthracoid animals buried during the fall or winter, the bacteridia will be likely to perish without giving origin to germs. But during the summer the conditions are almost always favorable for the production of spores, especially when the animal is buried at little depth, as is the rule. If you add that these germs may remain a long time on the soil without losing their virulence, and defile the herbage when heavy rains wash the dirt upon it, it will be easy to understand how the animals pastured there contract the disease from grazing this grass.

It is on record that at the farm of Roziers some sheep were attacked with splenic fever after eating the grass which grew over a pit where twelve years before some anthracoid animals had been buried.

Pasteur has also related the instance of a flock of 900 sheep, 400 of which died after having lain in their stable on earth brought there from a place where some cattle had been buried a great many years before.

Finally, in an epidemic of splenic fever in a village of Jura in 1879, twenty cattle succumbed in a few days and several of them were buried in a pit in a neighboring field. The year following Pasteur, Chamberland and Roux having discovered in the soil covering this pit anthracoid germs, fenced in the pit, and placed there four sheep. At the end of a fortnight these were dead, while the sheep feeding in the pasture around the enclosure and at some distance from it, continued in perfect health. Henceforth the etiology of charbon seemed to be established, and, as we shall see farther on, the experiments of inoculation have only confirmed these views of causation.

But before going farther, we must pause a moment to premise that not all animals are susceptible to charbon. If guinea-pigs, hares, horses, goats, cows take it, birds and fowls are refractory to it; other species, dogs, cats, carnivorous animals generally, if they are not altogether refractory, nevertheless die rarely after inoculation with the bacillus anthracis.

Man is not by any means exempt from this formidable affection. Every year butchers, leather dressers, tanners, dealers in skins, etc., after having handled the flesh or the pelts of anthracoid animals, die of malignant pustule, which is nothing but charbon, with a primary localization at the place of inoculation after a prick, or scratch with an instrument, ordinarily appearing on the hands or face. In Germany, however, anthrax has been observed in the human subject without malignant pustule, that is to say, men have taken the disease as sheep do, by introducing the bacteridia into their organism by the respiratory or digestive mucous membranes.

Nevertheless if you take into consideration that cattle dealers, but-

chers, veterinary surgeons, tanners, are constantly exposed to the action of the contagium, and that very often anthracoid meat is exposed for sale in the shambles (in farming districts and villages especially), one is forced to admit that with respect to the anthracoid infection, man is relatively refractory, and thus resembles the carnivora.

Why these differences in receptivity? It is well known that species vary in their aptitude to take contagious diseases; some being refractory to certain affections, while others readily fall a prey to them. But this is not all. In the same species all are not likely to contract charbon. Thus among cows some are easily infected, others are refractory; our sheep are prone to take anthrax, those of Algiers resist the contagion. To what is this difference due? Without doubt to the state of the liquids which bathe the cells of the organism, and to the vitality of the cells themselves. When you reflect that a slight change in the composition of the culture liquids suffices to prevent growth and development of the bacillus, you can readily understand the variable results which may attend the inoculation with virus of animals similar in appearance. Age, the kind of food, fatigue, etc., are so many causes which may make the organic environment vary and render it more or less suitable for the nurture of microbes, and the development of this or that disease. The organism opposes a greater or less physico-chemical resistance to the invasion of the microscopic beings which bring with them disaster and death—according to the condition of the organic media, according to the state of health or of disease, according to the quantity of the infection. In order to get a clear idea of the power which the constitution of these media has on the life of the bacteria, it suffices to recall to mind this fact: It is known that birds are refractory to charbon. It is known also that the normal bodily temperature of these vertebrates is notably superior to that of mammals, and even attains 108° F. in the gallinaceæ. Now, experience shows that at this temperature, bacteridia develop with difficulty. May this not be the cause of the resistance of fowls to charbon? If this conjecture is well founded, we ought to be able to give charbon to birds by depressing their temperature. The experiment always succeeds. It suffices to plunge into water at a temperature of 77° F. the feet of a fowl that has been inoculated with anthrax, and to bring the internal heat down to 98½° F., which is the temperature of animals capable of contracting anthrax, to see them perish in from 4 to 20 hours, every part of the organism being invaded by the pestilential bacilli.

But this is not all, and the counter proof has shown that if before invasion by the bacteridia, this fowl, whose blood has been dosed with anthrax virus and cooled, is taken from the water and natural warmth restored, it is seen speedily to regain its health and vigor. Conversely, if you take cold blooded animals, as frogs, and raise their temperature by plunging them into a bath of 95° F. you may communicate anthrax to them although they are refractory to it at their normal temperature.[1] These experiments throw a flood of light on what is called in pathology, the organic aptitude or receptivity.

But an objection to Pasteur's theory here presents itself. If the bacteridia alone are the active agents of anthracoid infection, how do they kill? Do they act mechanically[2] by obstructing the capillaries of the

[1] Paul Gibier, Soc. de Biol., July 1, 1882.
[2] As Toussaint pretends (Acad. des Sciences, 1880.,

lungs, of the intestines, of the parenchymata, and in producing emboli? This interpretation is little in harmony with cases of charbon where few bacteria or none at all have been found. Do they cause death, as Pasteur suggests, by absorbing from the blood of their victims the oxygen necessary for their respiration, thus depriving the anatomical elements of this vivifying agent? How does this doctrine harmonize with the observations of Toussaint relative to anthracoid vaccination; with those of Chauveau touching the inoculation of the sheep of Algiers; with those of Pasteur himself concerning the vaccine virus of fowl cholera and of anthrax? Must it be admitted that in these cases the attenuation of virus by heat [1] or by successive cultures,[2] enfeebles the activity of the bacillus and renders it incapable of struggling to advantage against the anatomical elements while at the same time modifying the organic medium and conferring immunity?

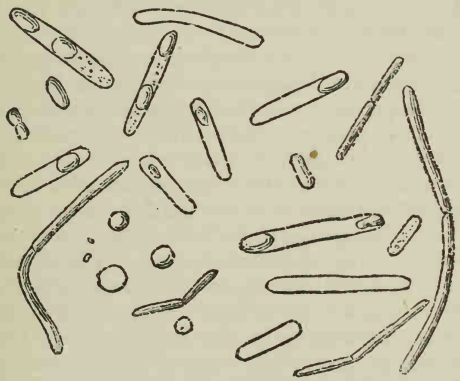

FIG. 22.—Organisms found in the blood of animals killed in apparent health, the blood being collected second hour after death. (Lewis.) × 1500.

Considering the uncertainty of these explanations, other theories have been put forth to explain the action of the bacteria in infectious diseases. According to some mycologists, the bacteria which are found in the blood of animals affected with anthracoid diseases, are only epiphenomena, that is to say, the bacteria develop only because the organism is already diseased. This hypothesis, which Robin [3] has always maintained, the substance of which is that the bacteria, vibriones, and microzymes multiply in the ratio of the alteration of the liquids, and are at best only the vehicles of the virulent humor with which they are impregnated, has been recently defended by Lewis.[4] This writer recalls the experiments of Brayell, of Bouley,[5] and of O. Bollinger [6] and of München, who showed that anthracoid blood containing no bacilli none the less communicates the disease when inoculated in other animals; also the experiments of Colin D'Alfort [7] who holds that experimental septicæmia begins before

[1] Toussaint.
[2] Pasteur.
[3] Traité des Humeurs, t. 2, p. 247, and Chauveau Art. Septicémie, in Dict. Encyclop. des Soc. Méd., t. viii. and ix., 3d series.
[4] *Loc. cit.*, p. 525, *et suiv.*
[5] Archives des Virchow, 1858.
[6] Zur Pathologie des Milzbrandes.
[7] Bul. de l'Acad., October, 1873.

bacteria are present; also his own observations, which seemed to prove that the blood of twenty rats, accidentally asphyxiated by privation of air, contained rods exactly like the anthrax bacillus; those also, of Signol, who found bacilli resembling those of splenic fever, in the blood of healthy animals asphyxiated by carbonic acid, and whose blood inoculated in sheep caused death without any microbe formation, while, moreover, the inoculated blood presented no trace of putrefaction. This writer invokes besides the fact of the *spirillum* of recurrent fever, which exists in the blood during the paroxysm, while it disappears during the interval. He remarks that if this microbe were the cause of the fever, it would not exist merely at the moment of the greatest intensity of the fever, but before, as well. It is more easy to admit that it only thrives and multiplies when the medium is favorable, that is to say, during the paroxysm. This objection, moreover, is easy of interpretation by Pasteur's theory, which does not ignore the fact that the microbes multiply, resist or die, according to the nature of the media.[1]

Lewis cites another fact which is of more importance. "It has been proved," he says, "that the living tissues of the body, in certain circumstances when they are excited by irritants purely chemical, such as a strong solution of iodine or of ammonia, secrete a liquid, which, when it is transmitted from one animal to another, is not less virulent than the exudation consecutive to the introduction into the organism of a substance swarming with bacilli. Observations on this subject have been published by numerous experimenters. Cunningham and I found a great number of bacteria in the blood of a dog killed by chemical poisons. These bacteria could not have been the cause of death; nor could they have come from the ammonia employed to produce the inflammation. It seems from these results that the living elements and tissues of the body have in the elaboration of septic poisons a much greater part than is generally assigned to them." This goes to confirm Gautier's observations on the production of the toxic ptomaines (cadaveric alkaloids), in the living and healthy organism, after Brouardel and Boutmy had found them in the cadaver.[2]

It remains to inquire if the bacteridia developed in the conditions of which Lewis speaks, may not have been carriers of the infectious or poisonous principle from the sick to the healthy organism?

Whether this may be so or not, Lewis concludes that the bacilli or spirilla "are only epiphenomena, the specific change of the liquids of the body taking place before the least trace of their presence can be discovered." This notion of a secretion of toxic principles by the cellular elements themselves in the healthy animal, is so like the one above stated, that we must briefly consider the latter, namely, the view which teaches that in the transmission of infectious diseases, the schizomycetes are only the vehicles of a morbid principle existing in the blood of the sick animals, which they concentrate in their organism, just as certain other vegetal or animal products are accumulated in the protoplasm of various cells. This hypothesis is in accord with the researches of Panum, as confirmed by Hiller, Bergmann, Heidenbaum, Wolff, Kussner and others,

[1] Signol, Compt. Rend. Acad. de Sc., t. lxxxi. Murchison, Brit. Med. Journ., April, 1879.

[2] A. Gautier, Acad. de Méd., June, 1881. Brouardel and Boutmy, French Association for the Advancement of Sciences, Congress of Rheims, 1880. In this connection the experiments of Vulpian on healthy human saliva are of interest. (Dogs inoculated with such saliva perished as by blood poisoning.)

on the transmission of septicæmia. Panum observed that the coagulum which an infected liquid produces on boiling, is more virulent than the liquid itself. The principal facts demonstrated by him may be summed up thus: 1. The perfectly clear liquid which is obtained by filtering putrefied solutions of animal substances through several thicknesses of filtering paper (charcoal, compressed wadding, porous pottery may be used instead), communicate the same infectious symptoms as the substance not filtered; 2. In boiling the liquid eleven hours its toxic properties are not altered; 3. Although an alcoholic extract of the liquid is innocuous, the virulent action of an aqueous extract of the same liquid is very intense. This is why Panum concludes that a liquid which may preserve its toxic properties after being filtered, boiled, evaporated to dryness, and whose residue has been treated by cold and by boiling alcohol, then dissolved anew and again filtered, cannot longer contain living organisms.

Facts equally conclusive have been pointed out by Dr. B. W. Richardson. This observer relates an instance where the sero-sanguineous liquid of the peritoneal cavity of a patient affected with pyæmia, communicated the disease to several animals successively; he also found that the septic poison which caused these disorders, was susceptible of combining with acids to form salts which preserved the infecting properties of the original substance. (Lancet, April 3, 1875, p. 490.) Bergmann succeeded in obtaining a similar substance, which when inoculated in very minute quantity provoked septicæmia, and to which he gave the name of *septine*.

Some of the propositions of Panum contradict the experiments of Chauveau, who proved that the virulence of infectious liquids is due to the solid particles of virus,[1] and to those of Pasteur which prove that a putrescible liquid, blood or lymph, filtered through a plaster filter in a vacuum and deprived of bacteria, may be inoculated without any effect.

At the same time, if the animal organism is capable of generating infectious principles—and the instances cited by Richardson and the facts set forth by Gautier and Vulpian seem indubitable,—it is not impossible that these principles may be assimilated by bacteria developed consecutively to the injection, which become then powerful agents of infection. As for the rapid development of the schizomycetes in the diseased organisms, may not certain morbid states of the blood and tissues be the *sine qua non* to their nutrition and multiplication?

It is evident that there still exists much that is obscure and hypothetical as to the rôle of the microphytes in the production of diseases. It seems clear that all the bacteria act the part of ferments; that there is a great variety of distinct species, though they cannot be morphologically specialized; and that the results of their vital action are multiple; it is almost certain that they act as the special ferments of the zymotic diseases, though we are ignorant of the rationale of their operations, or the differences in the results. Virulence and contagium become only another word for fermentation with the cryptogam parasites for ferment, and the animal organism for fermentable compound; the same holds good of the infectious diseases of vegetables, such as potatoes and cereals. In harmony with this germ theory, we must suppose that the organism of a dog is not *fermentable* to the contact of the variolic cryptogam, or of the anthrax bacteridium, in the same degree as that of the organism of men, of cattle, etc., is.

[1] He proved this by inoculations with filtered liquids (which proved inert), and with the solid (figured) elements, whose virulence was demonstrated.—Trans.

But this is not all; the pathogenic bacteria do not always find a suitable habitat in the fluids and tissues of animals naturally susceptible to them, and it would seem that for them to find a proper culture field there, the organism must be already morbid. Hence it is that you do not meet with bacteria at the period of the first chill of puerperal fever, of purulent infection or of urinous fever; they do not appear till later.

We are not more enlightened as to the reason why, having once invaded the body of an animal or man, they do not continue their work of multiplication and destruction till death ensues; the latter being perhaps the exception rather than the rule.

[In the discussion of Anthrax the author has neither done full justice to his subject nor to those eminent workers Greenfield and Koch, who have accomplished so much to elucidate the pathogeny of this affection. From the day when Pasteur reproduced the disease in healthy animals by the remote products of his cultures, which experimentation has been successfully repeated by Koch, and Pasteur, followed by Greenfield, demonstrated the immunity conferred on animals by attenuated virus, it was proved that medical science has command of the etiology of the disease, and that Pasteur's affirmation is true that *the bacillus anthracis is to charbon what the acarus is to scabies.* This fact must now be considered as settled, and *the accuracy of any experiments which contradict this conclusion must be challenged.*

One point established by Greenfield deserves to be mentioned here, as it tends to answer some of the objections which have been raised; viz., that there is danger of overrating the diagnostic value of anthrax bacilli in the blood, "for very frequently they are so limited to the affected regions that repeated examinations of the blood will fail to detect them in the earlier stages of the disease."—Trans.]

[**Swine Plague.**—There is a contagious disease which prevails among swine; and which has been very fatal to them in different parts of the world. Its ravages in the southern and western parts of the United States have been the subject of lengthy reports by Salmon and Detmers to the Department of Agriculture. Swine plague is a malignant pneumonia which causes certain characteristic changes in the lungs consisting in an infiltration with exudation, and finally hepatization of the pulmonary tissue; the lymphatic glands are always enlarged and the pleura is apt to participate in the inflammatory changes.

In 1876 Klein discovered certain micrococci in the blood of animals sick from splenic fever, which Salmon afterward identified as the cause of the disease. He demonstrated that this micrococcus exists in the blood during the life of the animal, that it can be cultivated in flasks, and that the sixth successive cultivation, made in considerable quantities of liquid, and which contained no other form than the micrococcus, still produced the disease. See Rep. Dep. Agriculture 1881-1882; also "Science" Feb. 8th, 1884, p. 157.—Trans.]

Fowl Cholera (Chicken Cholera).—Another infectious disease, *Fowl Cholera,* is also produced by a bacterium, a little micrococcus which is well cultivated, with conservation of all its virulent properties, in chicken broth made alkaline by potassa, but which dies when sown in infusion of yeast, although the latter perfectly suits the anthrax bacillus. This is an instance of the fact that the microbes do not develop in all liquids, and that a favorable medium is necessary, in accordance with the great law (applicable to all living beings) which was formulated by Lamarck and Charles Darwin.

The blood or the products of the culture of the blood of a fowl that has died of this disease, when inoculated in hares causes them to die rapidly, but guinea-pigs are much more refractory. The bacillus anthracis and the chicken cholera microbe are *aërobic*, developing in contact with air and not in a vacuum or in presence of carbonic acid.

Toussaint pretends that fowl cholera and experimental septicæmia are the same, for the virus vaccine of the one seems to confer immunity to fowls from the other. Pasteur, however, has rendered this conclusion improbable.

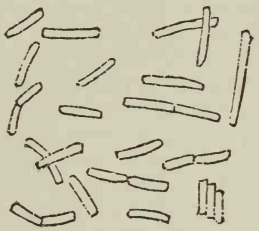

FIG. 23.—Organisms found in the blood of a mouse a few hours after death. (Lewis.)

Pebrine of Silk Worms.—Many years ago sericulture was in a deplorably languishing state; the worms died without spinning their cocoon. No one could discover the cause of the plague.

Lebert, Frey, Guérin-Meneville and Cornalia had indeed discovered that all the sick worms and moths contain a special microscopic parasite, but Filippi maintained that these "corpuscles" existed in all moths.

Things were in this state when Pasteur commenced his researches. He showed that the pebrine must be considered as due to the existence and development in the interior of the body of the silk-worm of a special parasite visible under the microscope, the *corpuscle*, (panhistophyton of Filippi). This parasite invades all the tissues and stifles them. Sick worms may transmit the infection to those that are healthy by the infliction of visible wounds with their claws, and by their excrement. The disease is then contagious, and rages the more the more thickly the worms are congregated together. It is also hereditary, for a diseased worm may communicate it to its offspring, in depositing the pebrine corpuscle in the eggs from which the latter are to spring. Every worm which, starting from a *corpusculous* egg, carries with it from the beginning the germ of the malady, dies before attaining the power of reproduction, and at the end of the breeding season all the chrysalides will have perished that came from infected eggs, and the living moths will have been poisoned by them; the pebrine germs will then pass into the eggs, ready to smite with the disease the next generation. Hence in order to suppress the pebrine, you must raise your silk-worms from healthy eggs. Pasteur's instructions in this regard have been most beneficial to sericulture; for in fact, he has not only proved beyond any doubt the parasitic nature of the pebrine, but he has indicated the sure means of prevention. He has also shown that the disease of silk-worms known as *flacherie* is due to a vibrio which lives in the alimentary canal of the worm, and arrests its digestion.

If you cause healthy worms to ingest a portion of the dejections of a sick worm, and see them perish in consequence with vibrios in their intestines, like those which they devoured, it is quite evident that it was

the ingestion of these ferments which caused the death. This is in fact what takes place. To prevent the contagion from spreading, it is essential, as Pasteur recommends, to breed the worms over a large extent of surface, and to keep them as far apart as possible.

The pebrine is hereditarily transmitted by the introduction into the egg of a corpuscle from a diseased parent. Heredity in the disease called *flacherie* is, so to speak, functional, manifesting itself by a weakness of the digestive tube, and a predisposition to allow inward fermentations to be established. In breeding silk-worms it is necessary to be very careful to eliminate all infected moths and chrysalides.

Fermentation of Urea. Ammoniacal Urine.—Every one knows that exposed to the air, urine decomposes. Rouelle in 1773, Cruikshank in 1798, Fourcroy and Vaquelin, Dumas and Muller, showed that this phenomenon takes place by the transformation of urea into carbonate of ammonia. Under what influence? Some maintained that it was a simple action of chemical decomposition. Pasteur and Van Tieghem pretend that it is a fermentation, linked to the presence of a torula, *micrococcus ureæ* (Cohn), which, in the urine of herbivora splits up into hippuric acid, benzoic acid, and glycollamine, by a simple change in the elements of water.

Murculus disputed these data; precipitating ammoniacal urine by alcohol, washing, drying and pulverizing the precipitate, he saw the urea undergo transformation into carbonate of ammonia: he concluded from this that the ferment was of a soluble nature and refused to assign to it the characters of a figured ferment.

Pasteur and Joubert parry the difficulty by the explanation that the transformation of urea into ammonium carbonate may be the result of a soluble ferment, but that this is fabricated by the torula.

But if this fermentation of urine is co-relative with the development of this torula, and if this torula does not originate by spontaneous generation, when the urine is ammoniacal in the bladder, there must then be ferment germs in the bladder. How did they get there?

The origin of the infection might seem clear when catheterism is practised, but how is it when no sound has been introduced to convey the germs? If, moreover, the germs are always present, why does not the urine oftener become ammoniacal? It is, however, known that not all urine ferments with the same readiness. Thus Feltz and Ritter introduced, by means of a sound, some ferment into the bladder of healthy animals, and the urine became ammoniacal only temporarily or not at all. This is because the urine of an animal in health is naturally acid, and the acidity prevents the development of the torula. On the other hand, if the urine accidentally becomes alkaline or neutral, or if the dose of the ferment is considerable and the urine little abundant, the torula may implant itself in the bladder permanently, and render the urine ammoniacal.

But, as Gosselin remarks,[1] the urine may become ammoniacal in the kidneys. In a patient whose urine as voided was ammoniacal, he drew off the urine by a catheter, and thoroughly cleansed the bladder with a carbolic injection; he then left a disinfected catheter in the bladder so that the urine as it trickled down from the kidneys might flow out by the sound. Now this urine was found to be ammoniacal as excreted by the kidney. It seems, then, that under special pathological conditions, and in

[1] Acad. de Méd., April 5 and 13, 1875.

particular, suppurative nephritis, the urine is akaline as it filters from the renal organ. As the urine never becomes ammoniacal without previous alteration of the urinary organs, one may be warranted in maintaining that the torula is an effect and not the cause.[1]

Experimental Septicæmia.

This affection is the result of a bacterium, the *Micrococcus septicus*, to which Cohn, Klebs and Eberth have attributed both *septicæmia* and *pyæmia*. It has been known since the experiments of Gaspard, of Flourens, and of Gunther, of Darcet, of Lebert, and of Sedillot in 1849, that pus put in contact with serous membranes or introduced into the veins, determines variable alterations, such as metastatic abscesses. Chauveau made a subcutaneous injection of putrid pus in a horse, and killed it; he made in another horse subcutaneous injections of pus coming from an acute phlegmon of the axilla of an old man, whereupon only phlegmons and abscesses of benign nature ensued. The difference is striking. Take a certain quantity of pus; strain and filter one portion, strain only another portion; the first, if injected subcutaneously shall give rise to no untoward result; the second shall provoke abscesses. Pus of a bad quality thus treated, occasions death only when injected without being filtered. If you filter and inject it, you will witness at the point of injection at the most only a little swelling. The experiment is quite conclusive; it is only the corpuscular parts which are toxic, the serum of pus is ordinarily innocuous.

It is not always so, however, for Panum and Zeulzer have shown that the toxic principle of liquids obtained by the maceration and putrefaction of fragments of muscle is soluble, resisting prolonged boiling and absolute alcohol; this *sepsine*, as Bergmann calls it, is then diffused in the serum, and the injection of it in the blood, deprived although this serum be of its figured elements, entails grave accidents, and even death in a few hours.

In examining under the microscope the secretion of wounds, Klebs discovered there the *micrococcus septicus*, the products of whose vegetation were credited by him with the power of forming thromboses and metastatic abscesses. He claims even to have met them in the interior of the organism in a case of suppurative osteomyelitis. May, then, these micrococci be considered as the origin of the septic phenomena? This would seem to be a warrantable conclusion, if with Tiegel you should filter a putrescent liquid of this kind, and see the unfiltered residue determine an intense septicæmia, when the filtrate produced only a temporary fever. The demonstration, to be complete, should include the production of the septic fever by the micrococci alone, and it should be shown that only these figured elements intercepted by the filter could have conveyed the infection, and this, as we have seen, is opposed to the experiments of Panum.

Orth, in his turn, in an epidemic of puerperal fever (puerperal metroperitonitis) at Bonn, in which there were no metastatic abscesses, injected in hares the sero-purulent peritoneal liquid containing the micrococcus, and witnessed the death of the animals. Bacteria were everywhere wanting, but the micrococci were found in the peritoneum, lymphatic vessels and blood of the hares. The human puerperal blood injected in its turn, and the blood of the hares that had died of the disease, produced in other animals the same phenomena.

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[1] *Vide* Rev. Mens. de Méd. et de Chir., October, 1877, and March, 1878.

Birsch-Hirschfeld, in studying under the microscope healthy pus and pus of bad quality, found that the second alone contained micrococci. The first was injected in animals with impunity; the second rapidly killed, and was the more fatal the more parasites it contained. At the necropsy, micrococci were found in abundance. When the pus contained bacteria, gangrenous phlegmons were noted and all the phenomena of putrid infection. According to this authority, the micrococcus develops pyæmia, the bacterium septicæmia.

According to Duclaux, also a pupil of Pasteur, the bacterium of pyæmia, which is both aërobic and anaërobic, is different from that of septicæmia, which is exclusively anaërobic. (Pasteur.)

It is the former which by its vegetation gives rise to metastatic abscesses. But Duclaux himself avows that purulent infection may exist apart from this vibrio, and even when it is dead, which proves that it is not to its development that pus poisoning is due. This is in accordance with the communication of Prof. Léon Le Fort to the Academy of Medicine in 1878, when the subject of purulent infection was discussed.

But does the blood of septicæmic animals always contain bacteria? Davaine is not positive on this point; Vulpian is more so. But it may be objected that the blood of a putrefying carcass swarming with bacteria, has been injected in large quantities with impunity. Onimus places on a dialyzer some septicæmic blood, and rests the dialyzer on distilled water; the latter is soon charged with organic products borrowed from the blood, and subsequently is full of bacteria. This liquid nevertheless remains inert when inoculated in an animal. Traube and Gschleiden inject into the jugular vein of a hare one cubic centimetre of a liquid containing bacteria, and at the end of from 24 to 48 hours they collect in a sterilized vessel a little of the arterial blood of the animal. This blood may be preserved for months if kept from all access of germs, without presenting any trace of putrefaction, a proof that the bacteria which they injected had been destroyed in the blood. Hiller went so far as even to inoculate himself with a liquid swarming with bacteria, and never experienced the least harm from it. But, say the advocates of the morbific microbe theory, this proves but one thing, namely, that there are bacteria and bacteria, some virulent, others innocuous, despite their morphological similarity, and, moreover, organisms are not always alike in their receptivity. There is no doubt about this last statement. The curious experiment of Chauveau on *bistournage* shows this. After the rupture subcutaneously of the spermatic cord [the testicle in the operation being twisted around the cord till all vascular connections are broken] the testicle of the animal, kept protected from contact with the air, remains free from all putrefaction although deprived of blood. It undergoes granulo-fatty degeneration, and ends in complete resorption. If, before practising bistournage, some serum rich in vibrios is injected into the jugular of the animal, or pus from putrid abscesses, the blood transports the germs into the organism, and then the dead testicle enters into putrefaction, when by its side the living testicle remains intact for some time. From this we see how much value should be attributed to the resistance of the organism in the presence of the infectious microbes. When the subject of an amputation is seized with septicæmia, and has at the same time a trifling lesion which would have otherwise escaped notice, do we not often see the latter suppurate?

May we not compare with cases of this kind the awakening of diatheses

under the influence of traumatisms, a phenomenon set forth in so clear a light by Prof. Verneuil? Nevertheless, according to Colin of Alfort, the virulence manifests itself before the appearance of bacteria in experimental septicæmia, and exists apart from them. Laborde produces septicæmia, and finds no microbes in the blood. What does this signify, reply the partisans of the germ theory, if not that the medium is not suitable for the life of these organisms? Look for them in other organs, and you will find them. In fact do we not know that the inoculation of vaccine virus, of the virus of symptomatic charbon, *in the blood*, does not determine morbid accidents, when the *subcutaneous inoculations* are always operative?

Let us return to septicæmia.

"In order that pus," says Chauveau, "introduced into the circulatory stream may determine pyæmic lesions, it is not sufficient that it be putrid, it must also have a sort of *specificity.*"

When a sheep, a cow, a horse, have succumbed to anthrax, and the blood is collected one or two days after death, this blood, when inoculated in guinea-pigs and hares, does not produce anthrax. The animals succumb to a new disease; their spleen is normal and their blood contains no bacteria. Inoculated, however, in other animals, this blood occasions death; sown in different liquids in contact with air, it gives rise to no microphyte productions. If, however, the blood be cultivated away from the air, in a vacuum, or in carbonic acid, it in twenty-four hours swarms with mobile and flexuous vibrios; you may then make successive series of cultures and a drop of these liquids reproduces septicæmia with its ordinary lesions. Experimental septicæmia is then the result of the septic vibrio. (Pasteur, Joubert, Chamberland and Roux.)

It is this disease which Jaillard and Leplat produced by inoculations with the blood of an anthracoid cow dead for some time, and as they found no microbes in the blood, they concluded that charbon might be transmitted without bacteria. Davaine proved that the disease studied by Jaillard and Leplat was not splenic fever. Pasteur showed that it was septicæmia.

But it seems that they were also mistaken in saying that in these cases the blood contains no microbes. In fact we have seen that cultures may be made of this blood in a vacuum or in carbonic acid, and an attentive microscopic examination will always show vibrios wriggling among the globules. What seems astonishing is that these vibrios, existing in so small quantity in the blood that their presence is sometimes doubtful, provoke the death of an animal in from twelve to fifteen hours. The reason that the followers of Pasteur give for this, is that the blood is not the favorite habitat of the septic vibrio. It is in the muscles and peritoneum that you must look for them; there they swarm by millions.

Another singular fact is worth mentioning. Signol has shown that it suffices to asphyxiate an animal in order that after fifteen or twenty hours, the blood of the venæ cavæ and of the heart shall become so virulent that if inoculated in sheep and hares, the latter die in from twenty to thirty-six hours. Signol pretends that he has thus communicated anthrax; Pasteur maintains that he has done nothing but provoke septicæmia. The germs of the septic vibrio are in fact very widely diffused and common. The intestinal canal contains them, and after the death of the animal they pass through the walls of the intestine, enter the blood and tissues and commence the work of putrefaction.

The septic vibrio is, then, an anaërobiotic animal. In contact with the air it is speedily killed. In order to undergo development, its corpuscle germs have need of the absence of air or the presence of carbonic acid. They then develop in from twenty-four to forty-eight hours, are like those of the bacillus anthracis, and may, like them, remain a long time in the air, in the water, or in the soil without being destroyed. They are very widely diffused, for they are present in all putrefactions, and if they do not smite us oftener, who are constantly surrounded by them, it is because they are not in the midst of conditions and media favorable for their development, and because the organism offers too much resistance to them. Nothing is easier than to diminish the virulence of the septic vibrio by changing the nutritive medium; but with a favorable medium its noxiousness is speedily realized. In the abdominal serosity and the muscles, it has the form of a mobile filament and is particularly virulent; in the blood it degenerates and its noxious character changes.

To sum up, we may say that septicæmia is putrefaction taking place in the living organism; but the multiplicity of septicæmias produced by Pasteur and Koch, does not allow us to admit without reserve a single septic bacterium, or to affirm that this affection has for sole cause a vibrio. Hence it is that it has been supposed that the offending substance might be an alkaloid (like the ptomaines for example), which induces a sort of putrefaction. (Cohnheim, Birsch-Hirschfeld, Hueter.)

[Septicæmia is a pathological condition characterized by putrid decomposition of the blood, pyæmia is a malady caused by the entrance of pus into the blood; there is not, however, entire agreement as to the definitive distinction between these two affections. Koch has produced in animals three kinds of septicæmia differing from each other in their pathoanatomical forms and in their microbes. The first is "malignant œdema," identical with Pasteur's septicæmia; it may be engendered by introducing vegetable mould into the peritoneal cavity of hares. The bacilli resemble those of anthrax, but they are never found in the blood. The second species, analogous to the septicæmia of Davaine, may be produced by the injection of putrid blood. The bacilli have an oval form with rounded ends. The third form, the septicæmia of mice, was produced by injecting putrid blood into the pleural cavity. The bacilli are thin, and abound in the blood inside of the white corpuscles. Pyæmia has also been produced by Koch by the injection of putrid matters; it is characterized by numerous cocci, isolated, or in the form of zoöglœa. Different kinds of cocci have been found by Rosenbach and Weichselbaum in pyæmia, and cultivated by them. Lastly there seems to be no doubt that septicæmia may be generated by animal poisons, apart from all presence of bacteria. The question can hardly be regarded as yet settled.—Trans.]

Puerperal Fever.—Puerperal fever is also a microbe disease according to Pasteur, Spillmann and Doleris; they have always found bacteria in the lochia of women affected with puerperal metroperitonitis. The appearance of these microbes in the lochial discharges, according to these authorities, always indicates with certainty the invasion of this dreadful disease. This affirmation needs confirmation; at any rate if true it does not prove that the bacteria are the cause of the disease. Léon Le Fort has brought to mind the fact that in a fatal case of pyæmia, and in another of septicæmia, Pasteur himself was not able to find or cultivate any special organism. No germs, no infection, is then not rigorously true.

[There seems, however, warrant for the belief that puerperal fever is

a form of septicæmia in which as causal factors, may predominate sometimes a chemical ferment, sometimes the septic vibrio and the round bacterium. (See the subject exhaustively discussed by Lusk in Pepper's System of Medicine, Lea Brothers and Co., 1885.)—Trans.]

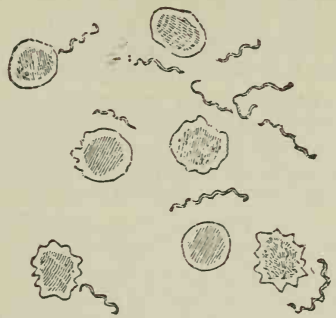

FIG. 24.—Spirilla found in the blood of certain patients. (Lewis.)

Symptomatic Charbon.—Splenic fever is the consequence of the evolution of the *bacillus anthracis;* symptomatic charbon, *Chabert's disease, emphysemato-gangrenous tumor,* is the result of a mobile microbe which pullulates in the connective and muscular tissues of the tumor, which is rare or even absent in the blood, which is retained by the porous porcelain filter, and which, inoculated in cattle or sheep, reproduces the same disease. The liquid which has traversed the filter is innocuous. The horse and the ass resist the invasion of this microbe, which introduced into the blood of sheep determines only an ephemeral fever, while in subcutaneous injection it reproduces the disease. We shall see farther on, that use is made of this peculiarity in instituting a preventive vaccine for this affection by the process of intravenous injection of the virus of Chabert's disease which gives immunity against the disease.

FIG. 25.—Spirillum, or Spirochætæ plicobile of Cohn. × 650.

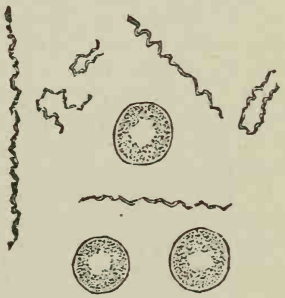

FIG. 26.—Obermeier's spirillum of recurrent fever. (Weigert.) × 660.

Intermittent Fever. Malaria.—A long time ago, Salisbury announced that intermittent fever was due to certain living organisms. He, however, gave no demonstrative proofs. Later, Balestra, Eklund, Safford, Bartlett, Archer, Burgellini, Griffini, Quinquaud and Magnin, noticed the existence of bacteria in malarial subjects. More recently Klebs and Tommasi-Crudelli, have described the *bacillus malariæ*, which really

Laveran and Richard had previously found, described, and delineated, in the blood of malarial patients in Algiers, under the form of spherical pigmented elements, of mobile filaments, etc., of the dimension of 8 to 9 μ., especially abundant in grave cases at the onset and during the paroxysm.[1] These microphytes disappear under the action of quinine treatment; a weak solution of quinine kills them rapidly on the object glass of the microscope. But may not these have been simple instances of alteration of the globules, hæmatoblasts and leucocytes? it has been asked.

In point of fact, knowing the scrupulous exactness and sagacity of Laveran, we can hardly doubt of the existence of these bodies in the humors, the spleen, etc., of the subjects of paludal fever; yet it must be confessed that the criterion of culture and inoculation is altogether wanting.

Nevertheless Klebs of Prague, Tommasi-Crudelli of Rome, report that they have succeeded by the inoculation of microbes, collected in those malarious countries, in giving intermittent fever to hares; but Cuboni and Marchafiava have not been able to obtain like results by inoculations of dogs. On the other hand, Orsi of Pavia affirms that he has found the bacillus malariæ in persons in good health.[2] Marchafiava also recalls the case of Bochmann, who after injecting under the skin of a healthy person the secretion from a labial herpes vesicle in a malarial patient, saw paroxysms of intermittent fever produced. Leoni in his turn reports similar success from two inoculations.

This parasite, which probably flits about in the air or water or inhabits the soil of marshy countries, has some claim to be considered as the cause of the febrile paroxysm, since it appears for the most part at the moment when the latter is about to come on or is actually in progress. Nevertheless it may also be legitimately maintained that the bacterium makes its appearance at this moment because the fluids of the economy are disturbed and have undergone a peculiar though unknown chemical modification favorable to the parasite's existence.

Recurrent Fever.—In recurrent fever the blood has been found infested with a *spirillum*. Virchow first, then Obermeier, pointed it out (1868-1872). This organism is from 6 μ. to 40 μ. in length; it is killed by a temperature of 65° C. (151° F.), by potassa, glycerine, and the salts of mercury. It is only during the febrile paroxysms that it is met with in the blood. Obermeier's attempts to inoculate it have not succeeded. Motschutkowsky says that although he has also failed in inoculations of animals, he has succeeded in inoculating human subjects with the blood of patients affected with recurrent fever, whether that blood contained *spirilla* or not. Engel and Lebert have vainly tried to inoculate relapsing typhus; Koch and Carter pretend nevertheless that they have transmitted the disease to monkeys. But we should remember that Vulpian has obtained similar results by inoculating hares with healthy human saliva.

Certain observers, as Manasseim, could not find the schizomycetes in the blood of certain febricitants. Birsch-Hirschfeld observed them two days after the crisis; Laskausky, in thirty-two cases, saw the microbes increase in the ratio of the temperature, while Heydenreich maintains that a high temperature tends to destroy them. Though seen by the before

[1] Revue Scientifique, April 29, 1882; Acad. des Sciences, February 20, 1882.
[2] Journal d'Hygiène, 1881, p. 383.

mentioned observers, by Engel, Weigert, Bliesener and Litten, they have not yet been cultivated. Are they the cause or the effect of the fever paroxysms?

Pneumo-enteritis (Typhoid Fever) of the Hog.—This disease differs from anthrax in that the fresh blood contains no foreign matter and is not infectious, but the blood at the end of a certain time gives by inoculation pneumo-enteritis, and thus assimilates this disease to septicæmia. The peritoneal liquid, also fragments of the parenchyma, provoke it.

Leisering in 1860 found in victims of this disease certain bacilli which he likens to the bacilli of anthrax. Klein affirms that the microscopic examination of culture liquids which reproduce the disease has proved that they are the habitat of a bacillus having all the characters of Cohn's spore-producing *bacillus subtilis*.

Diphtheria.—Henle in 1840, Buhl, Laboulbène, Tommasi, Hueter and Trendelenburg, have found micro-organisms in the false membranes of diphtheria. Cartel, in cases of diphtheria and croup, discovered a microbe, the *micrococcus diphthericus* (Cohn), which invades with its de-

FIG. 27.—Spirilla in chaplets found in the blood of patients sick with fever at Bombay. (Lewis.)

FIG. 28.—Bacillus of typhoid fever of the hog, cultivated in the aqueous humor of the hare. (Lewis.)

structive vegetation the diphtheritic exudations, the mucous membrane, and the lymphatic vessels of the pharynx, of the larynx, of the nares, the blood, the muscular tissue, the liver, the spleen, and even the bones. Eberth and Rassiloff have endeavored to show that in this so contagious and fatal affection, the micrococcus plays a causal part. To prove this, they inoculated the cornea of animals with products of the diphtheritic exudation, and they saw the micrococcus vegetate and invade the whole eye. But in these cases, as Dolchenskow has shown, if the cornea becomes diphtheritic and the hare dies from blood poisoning in four or five days, the same result may be obtained with bacteria from divers putrefactions. The experiments of Letzerich (in 1874) have not been more successful in settling this question. What may be truly said is that the cornea is a favorable medium for the development of the micrococcus, whose vegetation in a tissue may produce inflammatory reactions, embolic infarctions and necrosis. But is it certain that it is the micrococcus which is the principle of contagion? What seems to go counter to this hypothesis is the existence of cases of general diphtheria where the whole organism is poisoned from the start. In these conditions the fungus cannot have had the time to vegetate and produce its disorders. Nevertheless it is just to add that, Klebs and Talamon, by the inoculation of diphtheritic membranes in guinea-pigs, hares, pigeons and cats, have seen false membranes

develop in the throat of these animals. The future will no doubt clear up difficulties and explain the seeming contradictions which now beset this subject.

[In this country Wood and Formad have made extensive researches into the etiology of diphtheria; they constantly found micrococci, but saw no evidence that these were causal. The same micrococci appeared in false membranes produced by caustics, as ammonia, and even in the scrapings of the healthy human tongue. Heubner produced in rabbits non-specific false membranes by tying a ligature around the neck of the bladder, thus interrupting the circulation; a pseudo membrane in every respect resembling that of diphtheria was formed over the strangulated surface. Heubner's results agree with Jacobi's definition of diphtheria as "the compound of severe inflammation and necrosis."

Loeffler has lately published some experiments of his, in which he thinks that he has settled the question as to the relation of the micrococci of diphtheritic patches to the disease; they are in no sense causal, but are identical with those found with any infectious disease associated with lesions of mucous membranes. There are certain bacilli, however, first discovered by Klebs in 1883, which Loeffler constantly finds, and which seem to be something more than accidental complications. These bacilli, which are detected by a peculiar staining process (see Boston Med. and Surg. Jour., vol. cxii., p. 62), are found in the oldest part of the membrane, and penetrate farther toward the tissues than the other bacteria; products of their culture when carried to the twenty-fifth generation have produced the same effects on animals by inoculation as the diphtheritic virus. They are now called Klebs' bacilli; are motionless rods, partly straight, partly curved, about the length of the tubercle bacillus.—See Boston Med. and Surg. Jour. loc cit. Also Jacobi on Diphtheria, in vol. i. of Pepper's System of Medicine.—Tr.]

Virulent Diseases.

Small-pox. Vaccine Disease. Glanders. Sheep-pox. Rabies.—From what elements do the virulent humors derive their activity? Is it from the serum. Is it from the granulations which these liquids contain? Chauveau has shown that the more you dilute with water a virulent liquid, the less likely you are to see positive results follow your inoculation. If the active principle be something uniformly and chemically diffused through the liquid, one part would be as noxious as another, though the virulence would diminish in the ratio of the dilution. This is not, however, quite the case. It is found that all parts of the liquid are not equally impregnated with the virus, and, moreover, the latter when inoculated in however small a quantity, has the faculty of reproducing itself. It has in fact been sufficiently demonstrated that the virus is of a figured character, and that *contagion* is the function of solid corpuscles held in suspension in the virulent liquid. The more the liquid is diluted, the less abundant are these corpuscular elements in a given drop, but if there chance to be in this drop some of these solid particles, the inoculation succeeds, if not, it fails. This has been proved to be the case with small-pox and with vaccine matter.

Chauveau has gone even farther. It has been found that filtration, however carefully it may be performed, does not remove all the solid particles from serum, therefore Chauveau was obliged to resort to

the method of diffusion in order to separate his serum from the granulations. By carefully adding water to the virulent liquid, you have a rapid diffusion of all the soluble principles, but the solid elements settle to the bottom. If the virus were a soluble chemical ferment it would be equally diffused, and a drop of the supernatant liquid would have a certain infectiousness. This is not the case, you have got to obtain with your pipette the liquid from the lower stratum containing the granulations (which are not diffusible) before you obtain any result. These granulations Chauveau has separated from the infections secretions of glanders, he has isolated and washed them with pure water, and has always found them active and virulent.

It has not, however, been proved that these granulations are microbes; mycologists have not yet succeeded in cultivating them, and by inoculations, reproducing the disease.

Hence the faculty of virulence belongs to the solid particles suspended in the virus. Virulent diseases are transmitted by contact, which may be mediate or immediate. The atmosphere, then, does not contain the principles of contagion in the form of gaseous emanations, but in the form of solid corpuscles irregularly diffused. If the germs are abundant, many persons will be simultaneously attacked, and even aside from the immunity which many individuals possess, there will be some who will fall victims because they chance to meet with the contagium corpuscles, while others, more fortunate, will escape.

Sheep-pox.—The only figured particles which exist in the sheep-pox virus are the granular elements. It is among these that the noxious principle ought to be found. Now, if after having inoculated this virus in the sheep, you study under the microscope the resulting pustule, you observe the connective tissue undergoing proliferation, its plasmatic star-shaped elements multiplying, and masses of leucocytes forming. These elements are granular, but there is not a single free granulation in the liquid; nevertheless it is inoculable, and as virulent as the liquid which produced the pustule. Hallier and Cohn have deemed themselves warranted in pronouncing this affection a microphyte disease; meantime complete proof must be waited for.

Small-pox.—In 1865, Coze and Feltz discovered bacteria in the blood of a small-pox patient. They injected this blood into the veins of hares and communicated a grave and fatal disease; they found in the blood of the hares bacteria like those they had injected, and they drew this conclusion:—the hares died of small-pox, the bacteria were the cause. Now the disease communicated to the hares could not have been small-pox, for the good reason that this animal is absolutely refractory to it; if bacteria are the cause of variola, you ought to be able to find them in the blood and pustules of small-pox patients, but they are never to be seen there. However, Luginbühl and Weigert claim to have discovered in the small-pox pustule the granulations which we have mentioned as existing in sheep-pox, also certain micrococci which are found even in the tissues of the victim. But it must be admitted that these researches lack the experimental criterion of culture, and positive inoculation in a species capable of contracting small-pox.

Vaccine.—Keber of Dantzig was the first, in 1868, to call attention to certain microscopic organisms in vaccine matter. Chauveau, Burdon-Sanderson, Klebs and Cohn have described these micrococci under the name of *micrococcus vaccinæ*. (Cohn.) Cohn pretends to have seen these

micrococci develop in a drop of vaccine matter under the microscope at a temperature of 95° F., and he affirms their complete identity with the micrococcus of small-pox. Supposing these observations to be true, we still await the proof that this micrococcus is the cause of the disease.

Glanders.—Galtier (Acad. des Sc., Sept. 6, 1880) has announced that glanders is transmissible by inoculation from the horse to the hare and from the hare to the ass; the saliva of a glandered horse injected under the skin was found to communicate farcy to an ass; after fifteen days of complete desiccation the virus lost all its virulence. Is the contagion of glanders of a living nature? This has not yet been demonstrated.

[Since this article was written, satisfactory proof has been obtained that the septic principle of glanders is a bacillus discovered by Christol and Kiener, and figured by them as made up of a chain of nearly globular elements enclosed in a common sheath. In 1882 Bouchard, Capitan and Charrin cultivated these microbes in a neutralized extract of meat through five successive cultures, using in each case a milligram of the previous culture, or less than $\frac{1}{1000}$ part of the culture liquid. Counting that the milligram of pus would give to each centigram of the first culture liquid 1,000,000,000 bacilli, it follows that the second culture would on the principle of dilution, contain 1,000,000, the third 1,000, the fourth 1, while for the fifth it was as 999 to 1, so that it would receive nothing unless the germ were multiplied in the culture liquid. Inoculation of a cat with this fifth culture, started originally from a nasal ulcer of a glandered horse, led to a fatal result in twenty-five days, with suppurating tumor of the left testicle and inguinal glands. The products of the first cat were inoculated on the second, those of the latter on a third, those of the third on a guinea-pig, and those of the guinea-pig on an ass, producing in every case specific lesions of glanders, including miliary nodules and abscesses, and death respectively on the following days: 16, 7, 31 and 10.

In September, 1882, and the two succeeding months, a similar course of experiments was conducted by Schütz and Löfler at Berlin. The virulent matter used for starting the culture was procured from the pulmonary deposit and spleen of a glandered horse; the cultivation was continued through eight successive culture fluids. One horse was successfully inoculated with the product of the eighth culture, and a second with both the fifth and eighth. The first died on the fifty-eighth day, and the second, now very weak, was sacrificed on the fifty-ninth. Both showed the most extensive lesions of glanders alike in the skin, the lymphatic glands, the pituitary and laryngeal mucous membrane, and the lungs. To demonstrate the bacillus, they take a thin layer of the infecting liquid on a cover glass, dry it, stain with methyl violet, wash with dilute acetic acid, dehydrate by absolute alcohol, and clear by oil of cedar. Like other pathogenic microphytes, this may be preserved for months or years if thoroughly dried, but in the moist condition it is easily destroyed by heat (133° F.; Viborg, Hofacker, Renault), chlorine and the disinfectant chlorides and sulphites.[1]—Trans.]

Canine Rabies.—Klebs believed that he had found bacteria in the lymphatic glands of a rabid dog; but his observations have not been confirmed. That the germ is *particulate* is proved by its power of indefinite increase, also by Bert's experiment of filtering the saliva through plaster,

[1] (James Law, F.R.C.V.S., in Art. Glanders, in Pepper's System of Medicine, p. 912.)

only the solid residue left on the filter being virulent. (Law.) The bacteria found in the saliva of rabid dogs are doubtless ordinary septic bacteria.

[From the excellent treatise of Law on Hydrophobia (published this year)[1] we condense the following facts. "The point of election of the germ appears to be mainly the nervous tissue. Pasteur found the brain matter of rabid animals invariably infectious. He found that by direct inoculation in the brain substance the period of incubation was abridged, a few days sufficing to develop the disease. Pasteur assumes (as did Douboue in 1851) that the poison is propagated from the seat of inoculation to the brain through the medium of the nerves; this view, however, Law disputes, as not in harmony with the local inflammatory symptoms accompanying the inoculation, and heralding the general infection. The blood is not always infecting; Hertwig obtained rabies in only two cases out of eleven inoculations with the blood of rabid subjects. Galtier inoculated nine sheep and one goat by intravenous injection of the saliva of mad dogs, in no case with fatal results, but with the effect of fortifying the system, so that subsequent inoculation into the tissues of the saliva of rabid animals was harmless. Test inoculations made in the tissues of other animals with the same virus used in the intravenous injections, invariably determined rabies. Pasteur repeated these intravenous injections in dogs, with the result of rapidly inducing rabies. One of his animals recovered, and thenceforward resisted all further inoculation with the virus. Lussena of Italy in 1878 injected into the veins of two dogs, five grammes of blood taken from a physician who had died of hydrophobia. One died of rabies on the twenty-fourth day, the other at the end of 140 days. "These data," says Law, "do not warrant any very positive conclusion, yet they seem to imply that the receptivity on the part of the dog is greater than that of the small ruminants. They suggest further a greater relative potency, in the battle of life, of the blood globules of the small ruminants with this unknown rabific germ. This antagonism between the blood of the ruminant and the germ of rabies finds a parallel in the case of other disease poisons in their relation to the nuclei of the tissues. Thus animals may prove refractory to a small dose of the poison of anthrax, while a large dose will prove fatal. Talamon dilutes chicken cholera virus until it is non-fatal, though still affecting the system and conferring immunity from its attacks in the future. So with the lymph of ovine variola, which Peuch diluted to $\frac{1}{50}$ and injected, with the effect of producing slight fever, and immunity without vesiculation."

"More recently Pasteur has found that this virus when transmitted through several monkeys in succession becomes so weak as to be harmless to the animal inoculated, and yet protects the animal against the more virulent poison. This fact he utilizes by inoculating the mitigated ape virus in the brain of the animal just bitten, so as to render that refractory to the disease when the poison from the bitten wound shall reach it by its ordinary slow channel."

Pasteur's discoveries respecting the prevention of rabies by this mitigated virus, obtained by passing the canine poison through the body of the monkey, are among the most curious and important scientific acquisitions of the age.—Trans.]

Rubeola. Measles.—[It has been proved that the blood and nasal secretions, and even the tears, in this disease are inoculable. Howe demon-

[1] Pepper's Syst. of Am. Med., vol. i., p. 886.

strated this with morbillous blood (1757); Monroe, Locke and Katona with tears, nasal and bronchial mucus. Hallier supposed the *mucor mucedo* to be causal; Salisbury the straw fungus; William Pepper has proved both these experimenters to be mistaken. Coze and Feltz found *bacteria*, Klebs *micrococci* in the blood of morbillous patients, but they did not cultivate them. The specific microbe remains yet undiscovered.—Tr.]

Erysipelas.—Hueter and Orth were the first to advocate the microbiotic theory of erysipelas, which they had succeeded in communicating to hares by injecting subcutaneously liquid from the epidermic blebs of an erysipelatous patient. Lukomsky, who repeated these experiments, found in the erysipelatous parts the lymphatics stuffed with micrococci, and he affirmed with positiveness that the inoculations did not succeed except when the micrococcus was in the erysipelatous liquid. Vulpian and Trossier have found similar figured elements in the blood at the decline of erysipelas, and Tillmanns had five successful results out of twenty-seven inoculations.

This parasitic theory of erysipelas is in accord with a common opinion that this disease is never of internal origin, and that it is always due to some external infection, introduced through the nares, the lachrymal passages, by a traumatism, etc. More recently Dupeyrat[1] has confirmed the preceding observations, finding micrococci in necklaces or chains in the serosity of erysipelas (*bacterium punctatum*). In inoculating this serosity in hares, he has provoked death in eight days. He has cultivated this micrococcus in chicken broth with mineral salts, and obtained a liquid which produces erysipelas in hares.

But was it really erysipelas that was communicated to these hares? This affection is difficult to see on their skin. Moreover when the inoculation has been made in the living flesh, the disease produced was much more like septicæmia than erysipelas.

[Quite recently Fehlesen has found certain cocci (similar to, if not identical with those above described) in erysipelatous liquid; this coccus is always found in the connective tissue and in the interstices of the superficial tissues within the limits of the erysipelatous regions. Cultures of this bacterium develop rapidly in meat peptone; Fehlesen claims that with the product of these cultures he has produced genuine erysipelas in animals.

Prof. Cornil, the well known histologist (Med. Record, June 6th, 1885, p. 641), in undertaking to search for the microbe of erysipelas found a streptococcus, resembling very much that of phlegmon, but differing from the latter in that when inoculated, it always reproduces erysipelas. This remarkable fact is, according to Prof. Cornil, sufficient to affirm the existence of the micro-organism of erysipelas, in which it clearly stands in the relation of cause and effect. The microbes are of round shape, and are found generally associated in pairs, or in sinuous chains in the cutaneous tissue, in the lymphatic vessels, in the adipose cells of the skin and of the subcutaneous cellular tissue. These micrococci would seem to be identical with those which Dupeyrat and Lukomsky described. Whether the erysipelas be benign or grave, spontaneous or surgical, these mieroorganisms are always to be met with in this affection; they are sometimes conveyed along the general circulation, and excreted with the urine.

This microbe can be cultivated in gelatine, and inoculation with the product of culture gives rise to a simple erysipelatous erythema, without

[1] Thèse de Paris, 1881.

producing any severe accidents: and if the disease (which generally is but little dangerous) proves fatal in certain cases, as in surgical patients, or lying-in women, M. Cornil thinks that it is because the organism is not in these cases in a normal condition, also because these micro-organisms are found mingled with those of suppuration and of puerperal fever. The variations of degree of the severity of erysipelas may thus be explained by the addition of other bacteria to those which are special to this malady. (Paris Correspondent of the Med. Record.)—Tr.]

[**Typhoid Fever.**—Klebs (Philadelphia Medical Times, Dec. 3d, 1881) claims that he has proved "that there exists in typhoid fever a separate and distinct bacillus—the *bacillus typhosus;* that it undergoes certain transformations, consisting at first of little rods and small fine threads containing a spore in the centre and often at the end, which spores divide off and form new bacilli. It later assumes a larger thread-like form, twisted at the end, and frequently taking a beautiful spiral shape." He has also shown that "the bacilli are observed firs tin the masses of epithelial cells which accumulate in the alimentary tract, or in the air-passages; that they later penetrate the tissues and are carried along by the blood-vessels and the lymphatics, and form a large network among the tissues they invade; that under a certain procedure, which never causes this same staining in any other living organism or tissue, they appear of a blue color; that they are found only in enteric fever, in which disease every part of the human body is the seat of masses of these bacilli, their quantity corresponding exactly with the severity of the symptoms; and that they produce, when carried into the system of animals, exactly the same disease with the same morbid alterations as in men." He says further that "the bacillus typhosus enters the system by the respiratory passages and by the alimentary canal. This is the cause that in some cases of typhoid fever almost no abdominal symptoms are present, but a low form of pneumonia, developing from the very beginning, so that the lung seems alone to bear the brunt of the disease." He has found these bacilli in the greatest number in Peyer's patches.

Eberth (British Medical Journal, Nov. 26, 1881, from Virchow's Archiv. Bd. lxxxi. and lxxxiii.), has shown that in typhoid fever the intestinal mucous membrane, the mesenteric glands, and the spleen contain rod bacteria, differing, as he believes, from organisms found in the body in other conditions (among others in phthisis with extensive ulceration of the intestinal mucous membrane). In seventeen cases of typhoid these bacilli were found in six and wanting in eleven. In the six cases the number of bacilli were in inverse proportion to the duration of the disease. They are not found in the spleen in the cases of the longest duration, and only scantily in the mesenteric glands. These bacilli appear not to differ in shape and size from the ordinary rod bacteria, but Eberth believes that they differ from them in their small capacity for taking on the staining of hæmatoxylon, methyl violet and Bismarck brown.

The above statement (for which we are indebted to Hutchinson)[1] seems to represent the present status of the germ theory in its application to typhoid fever. It seems certain that all other microbes found in the dejections and diseased tissues in dothenenteritis, and signalled by various observers, are ordinary septic bacteria and have no causal relation, while it is probable that the bacilli of Klebs and Eberth are etiological, but further confirmation is wanted.—Tr.]

[1] Pepper's Syst. of Med., vol. i., p. 258.

Cholera.—[The belief in the microbiotic origin of cholera is of quite modern origin. Böhm in 1838 first found fungi in the dejections and in the intestine. In 1873 Lewis and Cunningham in India searched for characteristic organisms in cholera and cultivated those found. The bacteria and vibrios contained in stools and cultivated, proved to be common bacteria.

Last year Robert Koch was appointed to lead the German Commission for the investigation of cholera. The results of his work are now familiar to everybody. Nearly 100 cases came under observation, including the results of 60 autopsies made in India and Egypt. The dejections of thirty-two patients in India and of two at Toulon were examined. But little additional knowledge was obtained of the pathological anatomy of the disease. The alterations are limited to the small intestines, and are most marked in the vicinity of the ileo-cæcal valve; these consist of some degree of redness and swelling, with prominence of the solitary follicles and Peyer's patches. The microscope showed that this red margin corresponded to an invasion of bacteria, the now famous comma bacilli.

In a third series of cases the lower part of the ileum is of a dark reddish-brown color and the mucous membrane is studded with superficial hæmorrhages. Necrosis of the surface and diphtheritic patches of mucous membrane were seen in many instances. The comma bacilli were found in this series, and in addition various other bacteria, long thick rods, and very slender rods; the alterations were such as are found in necrotic and diphtheritic alterations of the intestinal tract and in typhoid ulcers when the tissue is first destroyed by pathogenic bacteria, and then invaded by other non-pathogenic forms. The comma bacilli always preceded the others and penetrated more deeply. The latter, though found in the intestinal contents, were absent from the blood and organs.

Koch claims that his comma bacillus presents definite characteristics which distinguish it from all other micro-organisms. It is identified by a process of staining similar to that used in the demonstration of the tubercle bacillus. A bit of the mucus from the intestine is spread smoothly on a cover glass and dried. The glass, after being thrice passed through an alcohol flame, is covered with a watery solution of fuchsine, then washed, and examined with a $\frac{1}{12}$-in. oil immersion lens and an Abbé's illuminator. Koch has succeeded in cultivating the comma bacillus in peptone gelatine. The comma bacilli thrive best between 86° F. and 104° F. Their increase is enormous within the first twenty-four hours, but after two or three days they begin to die. Koch states that when the transudation from the blood takes place into the intestine, the comma bacilli disappear, and are replaced by other bacteria, especially those of putrefaction.

Koch's cultures have been repeated by Nicati and Rietsch Pfeiffer, also by Van Ermengen and Doyen, who have confirmed his discoveries with reference to the appearances of the bacilli, and their behavior when cultivated. Babes has also lately made an extensive series of experiments which confirm Koch's statements. All these experimenters claim to have recently reproduced the disease in animals by the products of their cultures.

Koch, in reply to his critics, seems to have proved: 1. that the comma bacillus never appears in healthy individuals, in any part of the economy; 2. that it is a constant accompaniment of the cholera process, and is never found elsewhere. He did, however, while in India, detect these comma bacilli in a water tank used by the natives in the immediate vicinity of which a localized epidemic occurred.

Koch affirms that the comma bacilli are the cause of cholera, but he has not yet been able to substantiate this claim by inoculating the disease in animals, although, as above said, Doyen, Van Ermengen and others claim to have so done. Koch's argument is as follows (we quote from the excellent paper of Dr. R. H. Fitz of Boston, in the Boston Medical and Surgical Journal, Feb. 18 and 26, 1885, to which we are indebted for the concise epitome above given):—

1. They are constantly present in the disease, and nowhere else.

2. They are always found in immense quantities and usually as a pure culture on the linen defiled with dejections from cholera patients.

3. It is generally admitted that the disease is often transmitted through the soiled linen, and when such transmission occurs, the comma bacilli are the only organisms in question.

4. Their occurrence corresponds with the pathological changes in the body and the course of the disease.

5. The entire etiology of cholera is in harmony with the quantities of the comma bacilli.)—Tr.]

Yellow Fever.—[No specific micro-organism has yet been discovered, which may be accepted as sustaining a causal relation to this disease. A year ago, however, M. Domingos Frère, professor at Rio Janeiro, made in France a communication to the Academy of Medicine on the microbe of yellow fever. M. Frère claims to have discovered the specific microbe, to have cultivated it, and to have reproduced the disease in animals. He has also been able to produce an attenuated virus by which the disease is made to appear in a benign form, but giving effectual immunity from future attacks. Frère asserts that he has inoculated himself with this mitigated virus; a mild form of the disease ensued from which he soon recovered. He inoculated 500 persons under the order of the Emperor of Brazil; after going through the benign form of the disease, these persons were freely exposed to a focus of infection; the protective power of the mitigated virus was signally displayed. Frère's microbe is a cryptococcus. Bouley endorses his discovery; the attitude of the scientific world generally is one of suspension of verdict.]

Pneumonia.—[In Part II., Chaps. 9 and 10, of this work, will be found the arguments which are alleged in favor of the microbiotic origin of ordinary fibrinous pneumonia.]

[Scarlet Fever.—The London Med. Times and Gazette, Jan., 1882, contains an account of the supposed discovery of the scarlatinous microbe by Eklund, of Stockholm. He says that scarlet fever is rarely absent from the Swedish capital and from the barracks and dwellings on the isle of Skepsholm. In the urine of scarlatinous patients he has constantly found a prodigious number of discoid corpuscles, oval or round, their diameter being less than $\frac{1}{1000}$ of a millimetre, they are colorless or yellowish white, with cell wall and nucleus. They exhibit rotatory or oscillatory movements and multiply by fission. Eklund considers them to be schizomycetes, and proposes the name of *phlox scindens*. He asserts that he has found these same organisms in vast numbers in the soil and ground water of the isle of Skepsholm, and the greenish mould upon the walls of the old barracks where scarlet fever was most rife. He states that scarlet fever has occurred in children after drinking milk mixed with the ground water of the island, and he observed a case which followed immersion in one of the trenches of the island, and the drying of the clothes in a small room.

In another instance, scarlet fever broke out in a block immediately after exposure of the ground water by excavations. (Cited by Dr. J. Lewis Smith.) It is not claimed that any cultures were made of this microbe, or that any experiments on animals were performed with the virus.—Tr.]

Syphilis.—According to Donné, the pus of chancres contains the *vibrio lineola* of Müller, which is the only microbe capable of reproducing by inoculation the characteristic pustule. But this observation has not been confirmed, any more than that of Tigri concerning the bacteria of blennorrhagic pus. However, Klebs and Aufreest pretend to have found micrococci in syphilis which they have inoculated from man to animals. Bouchard also claims to show in his hospital service the microbe of syphilis. In blennorrhagia, Neiser and Bokai have discovered certain micro-organisms, and have even succeeded in inoculating this disease by the aid of cultures of the microbe.

Some recent researches of Martineau and Hamonic shed a new light on this question. Their report was made to the Acad. des Sciences, Sept. 11, 1882. According to this report, they excised an indurated chancre on April 29th of that year, and placed it in a sterilized flask containing Pasteur's culture broth. In a few hours a prodigious quantity of bacteridia developed as a grayish deposit at the bottom of the flask. They injected into the cellular tissue of the penis of a young hog a certain quantity of tiquid containing these bacteridia. The day following the injection, they noticed in the blood of the animal the presence of similar bacteridia. One month after the inoculation, certain cutaneous syphilitic manifestations (papulo-squamous syphilides) developed on the abdomen, while at the same time the hairs fell out. This experiment was checked by another, in which they inoculated a hog by pricking under the skin a needle charged with serum from an infecting chancre. Four days after this inoculation, examination of the blood of the animal revealed the existence of a bacterium similar to that of the first experiment. Fourteen days after, cutaneous syphilitic manifestations (papular syphilides) appeared on the abdomen. At first isolated, they became generalized, lasted for several days, then disappeared completely two months after the inoculation.

It would seem that if it was true syphilis that was produced in these animals, the evolution of this disease is much more rapid in hogs than in man, and that the constitutional disease does not present itself in the former with the entire series of accidents which appertain to human syphilis. All animals do not seem susceptible of contracting syphilis. It may even be said that the greater part are refractory to the syphilitic contagion, and that those which have been considered as having a certain aptitude present more or less resistance to the virus. In the hog it is notably so; if it be true that the syphilitic virus may be inoculated directly from man to this animal, it is none the less true that it is not inoculable from one hog to another, or from the hog to the monkey. This non-transmissibility from animal to animal appears moreover to be in relation with the rapid evolution of the syphilitic manifestations, with the rapid disappearance of the bacteridia and with the speedy recovery from the constitutional disease.

This difficulty in the transmission of syphilis from animal to animal explains moreover the rareness of this disease outside of the human species, and the difficulty which experimenters find in the solution of the new problems which it raises.

Diarrhœa and Dysentery.—Bacteria in abundance may be found

in the dejections of persons affected with diarrhœa and dysentery, but thus far nothing specific has been proved to pertain to any of them.

Tuberculosis.—[A long time ago Villemin affirmed the contagious nature of tuberculosis and pretended to have inoculated it in guinea-pigs. Tappenier communicated tuberculosis to hares and guinea-pigs by causing them to inhale the dried pulverized sputa of tuberculous subjects. More recently Toussaint succeeded in inoculating cats, pigs, etc., by causing them to eat tuberculous matter, also by inoculating them with the blood and secretions of diseased subjects. Klebs (1875), Buhl (1873), Wolff (1875), and Schuller discovered in tuberculous products an organism to which they gave the name *monas tuberculosum*. Reinsladler, a pupil of Klebs, claims to have cultivated Klebs' bacterium, to have inoculated it, and reproduced the disease, but with the first cultures. Klebs attributed to it the form of extremely minute monads, animated with very quick movements; Toussaint believed the infecting agent to be an immobile micrococcus. Both, however, pretend to have cultivated and inoculated these microbes, and to have produced tuberculosis by these inoculations.

To Koch, however, belongs the glory of having discovered the veritable microbe of tuberculosis. His investigations, cultures and inoculations are now known to the whole scientific world. His reputation as a painstaking, careful and conscientious experimenter cannot be gainsaid.

April 10, 1882, there appeared in the Berlinir Klinische Wochenschrift one of the first of those remarkable epoch-making communications from Koch, in which the microphyte of tuberculosis is described and the results of his cultures on dried gelatine. This microbe is a *bacillus*, now known as *Koch's bacillus*, the *bacillus tuberculosis*, which is readily detected in the sputa of tuberculous subjects and in tuberculous matter by a peculiar process of staining. Koch makes a staining liquid as follows: to 200 cubic centimetres of water, add one cub. cent. of a concentrated alcoholic solution of methyl violet, and two cub. cent. of a 10 per ct. solution of caustic potassa. When the mixture has been allowed to stand for several days and has become limpid, you place in it your thin sections of tubercle, and allow them to remain there for twenty-four hours.

The section is then mounted on a suitable glass slide, and covered with a thin glass cover over which had been previously placed a drop of a filtered aqueous solution of vesuvin. The preparation is then entirely brown with the exception of the bacteria, which are colored blue. The sections are more clear if treated with a little essence of cloves. The tubercle bacillus is stained by the other aniline dyes, but not by vesuvin. The specific bacteria are thin, and tapering at their two extremities. Their length does not equal that of one fourth the diameter of a blood corpuscle. Wherever the tuberculous process is at its commencement, these bacilli are found in large quantities, in groups, but they disappear when the process is about terminated. They are found frequently in the giant cells of tubercles. (lungs, spleen, liver, glands, articular fungosities of white swellings). The contents of cavities contain these bacteria in abundance along with other non-specific bacteria. Koch has noted in the hog, monkey, guinea-pig and hare the same facts as in man. He has cultivated the bacillus in the thoroughly sterilized serum of beef blood, and in dried gelatine, and with the products of his culture has inoculated various animals and reproduced the disease with all its characteristic lesions. In guinea-pigs and hares the product of the fiftieth culture when inoculated in the anterior chamber of the eye and in the peritoneum has generated tubercle; in

these cases so minute was the quantity and so far removed from the original infection that it would seem impossible that anything but the bacillus could have been operative in the production of the tubercles.

Various experimenters having at different times affirmed that inoculation with inert non-specific matters (such as silicon, bits of glass, gypsum, etc.), might generate true tuberculosis, Sternberg, by a convincing series of experiments (Am. Jour. Med. Sc., Oct., 1884), has shown that the lesions produced by such inoculations are simple inflammatory lesions, from irritation of the foreign particles, and are not transmissible from animal to animal, and that nothing but tubercle or the products of tubercle, can by inoculation produce tuberculosis.

After inoculation of the guinea-pig with a minute portion of the cultivated bacillus of the 10th to 50th or even 100th generation, after about eight days there appears at the point of inoculation a little nodule, then supervenes progressive invasion of the organs and tissues by the tuberculous process commencing at the first focus of infection, and death takes place in from five to six weeks. Prof. G. Sée has within the past year published a work on Pulmonary Phthisis in which he takes his point of departure from Koch's bacillus, as being the true and only cause of the disease. This new etiological conception has been fruitful in clearing up obscurities which have heretofore existed as to the nature of tubercle, which can now be identified and distinguished by its constant voucher, the bacillus; it has also enabled pathologists to comprehend in one generic unity acute and chronic phthisis, tuberculous meningitis, caseous pneumonia, scrofulous adenitis, osteitis, and lupus.—Trans.]

Dental Caries.—Observers have long signalized the presence of the *leptothrix* in the tartar of teeth, and some have even attributed dental caries to this fungus.

Some authorities, moreover, have assigned a microbiotic origin to *influenza* (see Part First of this work), *pertussis, scurvy, goître,* and *mumps.* If these be microphytic diseases, the specific microbe is yet to be discovered. Some physiologists have even affirmed that digestion is the work of the *amylo-bacter* (Van Tieghem's parasite); Hallier and Béchamp have pretended that the saccharifying action of saliva is due, not to ptyaline, but to a *leptothrix,* or to *microzymes;* that the pancreatic digestion is effected by an *anaërobiotic bacterium.* But the observations of A. Gautier, which have annihilated the microzymes, have disposed of these pretensions.

[**Malignant Endocarditis.**—The present theory of acute endocarditis is that it is in all its forms a mycotic disease; the local and constitutional effects being produced by the growth on the valves, and the transference to distant parts, of microbes, which vary in character with the disease in which they develop. The constant presence of micro-organisms has been proved; they are invariable constituents of the vegetative outgrowths; they are described by Osler and others as micrococci in chaplets; they bear a close resemblance and relation to those seen in puerperal peritonitis, of which in fact ulcerative endocarditis is a frequent complication.

In the way of experimental investigation of the properties of the micrococci not much has yet been done of a satisfactory nature. No conclusive culture experiments have yet been made. (Gulstonian Lecture on Malignant Endocarditis by Dr. Wm. Osler, 1885.)—Tr.]

MEANS PROPER FOR COMBATING THE VEGETATION OF BACTERIA. PREVENTIVE VACCINATIONS. ANTISEPTICS IN DISEASES SUPPOSED TO BE OF MICROBIOTIC ORIGIN.

With the microbiotic theory of infectious diseases, has arisen quite a new mode of treatment. Since infection and microbes are the same thing, if you destroy the latter you will cut short the former.

This is what Professor Alphonse Guérin has attempted to do with his cotton-wool dressing, which arrests the virulent germs of the hospital wards and prevents them from gaining access to wounds; this is also what Lister has accomplished with his *antiseptic dressing*.

It was, says Tyndall, the action of bacteria on wounds and abscesses which so often changed our hospitals into veritable charnel houses, and it is their destruction by the antiseptic system which enables us in our day to perform operations which a few years ago no surgeon would have dared to attempt.

Now it is known that the septic vibrio is an anaërobiotic bacterium. It cannot then develop septicæmia in a wound treated in an open manner, since it cannot live in free air. But if there happens to be an anfractuosity in this wound, the septic germs may then undergo development and invade the organism, communicating to it septicæmia. From this fact we can understand why wounds treated in an open manner do about as well as those treated with the most thorough antiseptic precautions, the conditions for the entrance of the septic vibrio seldom being favorable. Nevertheless it must be admitted that at the present day surgeons perform capital operations with much less danger, (thanks to the antiseptic spray and antiseptic dressings); opening with boldness and relative safety joints and closed cavities, (the abdomen, the thorax); that they more easily obtain union by the first intention in amputations; that erysipelas and the purulent infections are almost banished from our hospital wards. At the same time certain eminent surgeons contend that these results are not due to the antiseptic atmosphere, nor to the Listerian dressings, *quoad* protection from germs, but to the superior care and attention bestowed on the dressings with respect to sutures and drainage, and the curative actions of the so-called antiseptic liquids, phenic acid, alcohol, choral, etc., which beneficially influence wounds by preventing the tendency to molecular decomposition of the tissues and liquids rather than by hindering the development of, or destroying the bacteria, for the latter are still to be met under the carbolized dressings.[1]

If statistics of the major amputations treated by antiseptics give a mortality of 25 per cent. (Lister), of 8 per cent. (Volkman), those of Nelaton, who used simply alcohol dressing, give but 20 per cent., and those of Rose, who, to use Prof. Léon Le Fort's expression, leaves his amputation wounds exposed to all the germs of Switzerland, 17 per cent.

Nepven has studied the rôle of bacteria in surgery. He ranges them into three categories: *cocci*, *bacteria*, and *cocco-bacteria*. The first two species he says may undergo transformation the one into the other; the third is formed by the union of the two others. These organisms do not constitute the septic poison, he adds, but whether they produce the virus by fermentation, or whether, owing to their ready development in de-

[1] *Vide* Discussions at the Soc. de Chir. and Acad. de Méd., in Feb. and March, 1878–1879.

composing liquids, they charge themselves with the putrid poison, fix it and become its propagators, there is a dominant interest in opposing them.

But is carbolic acid as employed capable of destroying the germs of the septic vibrio, or micrococcus, which provokes the purulent infection? What gives the negation to this question, as we have just said, is that under the antiseptic dressings bacteria may be found active.[1]

In anthrax, in rabies, you cauterize the pustule or the bite with the red-hot iron, nitric acid, or caustic potash, bichloride of mercury, and these are agents the most destructive of bacteria. They cannot be too early employed, for the absorption of virus takes place with great rapidity. The first thing to do in such cases is immediately to apply a ligature above the wound, and to obtain surgical assistance. Sucking the wound (which is very hazardous, especially if there be any abrasion about the lips or mouth) has probably saved persons from death; this mode of treatment, which requires no little self-abnegation on the part of the attendant, may be resorted to if caustics cannot be obtained.

Davaine has experimented with reference to determining the effects of cauterization in preventing absorption of virus from wounds; his results are more encouraging than those obtained in similar trials by Rénault and Colin d'Alfort. One, two, and even three hours after depositing very virulent anthrax matter in wounds, he has thoroughly cauterized these wounds, and has seen seven out of ten of his animals escape the infection. Rodet has obtained the same results as Davaine, although, like Rénault and Colin, he made his inoculations with the lancet. It is presumable that if early and effectually performed, cauterization will guarantee protection.

In internal diseases it is quite legitimate to attempt antiseptic medication, though practically not easily realized, for there are great difficulties to be encountered in this direction; the antiseptics capable of killing bacteria in the organism are as a rule poisons to the anatomical elements of the tissues. However, it would seem that empiricism has found in quinine the antidote to the *bacillus malariæ*.

Bouchard has of late proposed to treat typhoid fever with a mixture of iodoform and charcoal with a view to local antisepsis, while Wilson and Bartholow have instituted the iodine and calomel treatment of this disease with the same end; certain American practitioners have advised the systematic treatment of diphtheria by calomel—small doses frequently repeated—with germicide intent, and have claimed good results. It is also believed by many therapeutists that the good effects of the iodides, and especially of mercurials, in syphilis, are owing to the fact of these medicaments being antagonistic to the microbes of the disease. It cannot, however, be said as yet that such considerations have anything more than a speculative value.

Sulphurous acid being an energetic antagonist of fermentation has been recommended in cases of septicæmia, of purulent infection, of glanders, of pulmonary tuberculosis with foci of purulent absorption, etc., in the form of sulphite of magnesia, or of hyphosulphite of soda and of lime.[2] Dujardin Beaumetz and Hirn have noticed that chloral prevents the development of putrid fermentation, but they

[1] Acad. de Sc., Note of Demarquay, Aug., 1874. Gosselin, Acad. de Méd., Feb. 1878.

[2] Pietra Santa, *Diseases due to morbific ferments;* treatment by hyposulphites and alkaline sulphites. Paris, O. Doin, 1875.

have seen it fail in the experimental septicæmia of the hare. Dujardin Beaumetz and Nocard have called attention to the curious property which waldivine possesses of mitigating the paroxysms of rabies; unhappily it does not cure hydrophobia. Denis Dumont claims to have cured rabies by pilocarpine, but the criterion of inoculation of the saliva was not resorted to; moreover G. Sée and D. Beaumetz had signally failed in their attempts to cure hydrophobia by this medicament. May not the method of attenuation of virus lead to prophylactic applications which in rendering the dog invulnerable to rabies, may stamp out this terrible malady? It seems as if to day, since Pasteur's brilliant experimentation, now so widely known, we are almost warranted in giving an affirmative answer to this question.

As for the germicides which may destroy, with safety to the organism, the bacteria of diphtheria, of small-pox, of measles, of septicæmia, etc., we are still ignorant whether there be any such. At the same time we are inclined to give in our unqualified adhesion to the doctrine which makes of figured ferments the causal factors in all communicable diseases, and we cannot but express our hope and our conviction that ere long therapeutics will be revolutionized by discoveries which shall do for all the infectious and contagious diseases of man what Pasteur's "mitigated virus" has done for charbon and chicken cholera, and that we shall then be in condition to rid mankind of those fearful zymotic scourges. So might it be!

With regard to febrile communicable diseases of which the active principle is probably a microphyte, internal antiseptic medication has as yet done little besides mitigating certain manifestations. Thus Vulpian with large doses of salicylic acid (ʒ jss a day); Desplats with salicylate of bismuth, (ʒ ij a day); G. Sée with quinine (gr. xlv a day), and more recent experimenters with antipyrine (gr. xlv a day), have obtained a constant and marked fall in the temperature of typhoid fever, with amelioration of the general condition, but the morbid cycle has not been a whit abridged thereby.[1]

It is not so with certain virulent affections whose microphytic origin is known or not doubtful. Every one is familiar with the immunity conferred from small pox by vaccination. Pasteur's preventive inoculations against chicken cholera and splenic fever are of a similar nature.

From all time in epidemics, observers have noticed in persons smitten a great difference in the intensity of the disease; some succumb rapidly, others have only a benign form of the affection from which they speedily recover. So also at the end of epidemics, it has been noticed that the cases which occur are mostly benign, which proves that the epidemic has lost its virulence.

Moreover, it is equally well known for centuries, that individuals who have suffered from one attack of a contagious disease are generally preserved from a second attack, that is to say, these diseases are not recurrent. Of this nature are typhoid fever, small pox, scarlatina, and measles.

Hence the happy idea has occurred to experimental therapeutists to attempt the inoculation of the benign form in order to preserve individuals from the grave form.

Hence it was that in the last century *variolization* was practised for

[1] Acad. de Méd., August, 1882. Desplats in Bull. Gén. de Thér. 1883; G. Sée, On the Treatment of Typhoid Fever, Paris, 1884; Arduin, on Antipyrine, Bull. Gén. de Thér. 1885.

small pox, virus being taken from a person affected with a light form of the disease—an operation sometimes, however, attended with fatal consequences.

Peuch seems recently to have had success in the inoculation of sheep with the dilute virus of sheep pox, immunity from the fever and dangerous local accidents being thus obtained. Every one knows, moreover, of Auzias Turenne's attempts in the way of *syphilization* to produce a milder form of venereal disease.

As soon as it was established that certain contagious diseases have microbes for their cause, it was natural to ask if the virus of each was always identical with itself, having always the same properties.

Coze and Feltz and Davaine had already remarked that certain kinds of virus, as that of septicæmia, augment in virulence in passing through the organism of animals; conversely, as we have before said, in certain media, such as meat broth, the same septic vibrio loses little by little its virulence. It is, then, evident that according to the media where they live, the micro-organisms undergo important modifications. Nägeli even thinks that in cases where an infectious disease breaks out spontaneously (and no one can doubt that instances of this kind sometimes occur, where it is impossible to trace the inception of the disease to contagion from an infected individual), the fungi of contagion arise from transformation of the ordinary non-infectious schizomycetes, or those of putrefaction, and this by reason of influences from the air and soil as yet unknown. He bases his opinion on this fact: the schizomycetes do not thrive in a nutritive liquid containing two per cent. of acetic acid; but if you sow in this liquid some *mucorinées* or *saccharomycetes*, these latter consume the acid and render the liquid fit for the schizomycetes, which henceforth appear in innumerable numbers.

Hence it is that in the juice of grapes or other fruits there develop first the saccharomycetes, which change the sugar into alcohol, and prepare the nutritive soil for the fungi of the flower and mother of vinegar which oxidize the alcohol into acetic acid. The liquid is then favorable for the mould fungi, which employ the acids for their nourishment and render the liquid neutral, so that the bacteria of putrefaction may commence their work. So, according to the medium, the ordinary schizomycetes (so Nägeli thinks) may undergo transformation into the fungi of contagion, and conversely. This clearly results from the fact that every disease has a commencement, and that at this moment the form of microbe peculiar to it must have sprung from some other form of micro-organism. We have, moreover, certain instances of the spontaneous appearance of infectious diseases and their fungi, which in a given circle manifest themselves endemically, and away from every epidemic area. For cholera, a like circle of endemic diffusion is found around Calcutta, whence the disease radiates under the form of an epidemic to India, Asia and Europe. Typhus is endemic in Central Europe and limited to certain populous cities, such as Munich.

No form of schizomycetes, according to Nägeli, remains the same unless it lives constantly in the same conditions. The faculty possessed by the milk schizomycetes of changing sugar of milk into lactic acid may be enfeebled and even annihilated when the milk is exposed to a high temperature or when these fungi are cultivated in other less favorable media. There is, moreover, reason to believe that by successive cultures one may convert the *anthrax bacillus* into the *hay bacillus*, from which it is in fact indistinguishable.

In these conditions of different vitality of the microbes, Chauveau, Toussaint and Pasteur have discovered the method of preventive vaccinations which confer immunity from their respective contagion. Chauveau accomplishes this end by minute quantities of the virus. The sheep of Algiers are very refractory to anthrax, but only to a certain extent; inoculations with great quantities of the virus kill them, while small quantities have but insignificant effects and confer perfect immunity against larger inoculation, as well as against the infection generally. Now Chauveau has seen the native sheep which are so susceptible to charbon killed by intravenous inoculations;—he prefers this method because he has generally proved in his experiments on vaccine and bovine peripneumonia, that certain viruses introduced in this way act with less energy, and none the less surely confer immunity. Chauveau, we repeat, has seen native sheep killed by the intravenous injection of one cubic centimetre of anthracoid blood containing 1,000 bacilli, when 100 bacilli caused no grave accidents whatever while conferring complete immunity. He obtained similar results with the virus of symptomatic charbon.

More recently, in employing virus dried, diluted and heated to near the boiling point for six hours, Arloing, Cornevin and Thomas have succeeded in successfully vaccinating against symptomatic charbon.

Toussaint, in heating defibrinated anthrax blood up to 132° F. for ten minutes, obtains a fluid which when inoculated not only does not kill the animal with anthrax, but confers on it immunity and renders it refractory to the most virulent subsequent inoculations.

Chauveau, in heating anthrax blood to 125° F. for about twelve minutes in capillary tubes, obtains an excellent vaccine virus. If the inoculation fluid be heated only to 120°F. it is necessary to prolong the heating to eighteen minutes.[1]

Pasteur has attained the same results with the virus of fowl cholera and of charbon by the method of cultures. The little micrococcus that causes fowl cholera is cultivated in chicken broth exposed to the air, at first keeping all its virulence; at the end of a certain time, however, if you test its virulence, you will see that this is much diminished, and after four or five months the liquid is no longer capable of causing the death of fowls, which suffer only slight symptoms. Now all fowls inoculated with this liquid attenuated in its virulence are thereafter incapable of contracting the cholera; they have been *vaccinated* against it. The cause of the diminution of virulence is the oxygen of the air in the flask, for if the same culture is carried on in a vacuum, the virulence continues unabated, and inoculations with this virus are fatal. Scarcely had Pasteur arrived at this result, when he obtained a still more astonishing success with the virus of anthrax, a disease which was yearly entailing on our agricultural interests a loss of millions of francs.

In order that the bacteridia may not give rise to germs which shall fix the virulence of the culture fluid, this savant has shown that it suffices to sow anthracoid blood in broth at a temperature of from 108° to 110° F. At the limit of this temperature the bacteridia live and reproduce themselves, but they never form germs. Thereupon, on testing the virulence of the flask after 6, 8, 10, 15 days, the same results are obtained as with the virus of chicken cholera. The culture which at the start

[1] Acad. de Sciences, June 26th, 1882. Also Arloing, Cornevin and Thomas, on the inoculation of symptomatic anthrax by intra-venous injections, and the immunity procured thereby. Acad des Sc., Nov. 2, 1880.

would kill ten sheep out of ten, at the end of five days would kill only five out of ten, and after ten days would cease to be fatal, only communicating a benign disease, which thereafter gave preservation against the malignant anthrax. Science was now in possession of a remedy against an unspeakable calamity to agriculture.

Since then public tests were made before the Society of Agriculture of Melun; 25 sheep and 8 cows were vaccinated, 25 sheep and 5 cows were taken as check tests and were not vaccinated. All were inoculated with a very virulent anthracoid virus; the 33 vaccinated animals suffered no serious effect from the inoculation; of the 30 not vaccinated, the 25 sheep died in 48 hours and the 5 cows were very sick. The success was complete. Since this event, 130,000 sheep and 20,000 cattle have been vaccinated. In the month of September last like experiments were made before the representatives of the government of Austro-Hungary, by a pupil of Pasteur, Thuiller, and they were successfully repeated at Berlin before a commission nominated by the German government. The trials made in Austro-Hungary have established the fact that the mortality from anthrax is from 90 to 94 per cent. in sheep not vaccinated, against 3 and 8 per cent. in those vaccinated. Pasteur made use of these figures in his reply to Koch and his partisans, who had violently attacked his method; he again referred to them in replying to Koch in person at the Congress of Hygiène of Geneva in September, 1882.

Greenfield, in England, has performed a similar series of protective inoculations against anthrax. He obtained the attenuation of the virus by cultivating it in animals smaller than sheep, as well as by culture in nutritive liquids.

Moreover Cosson (Acad. des Sc., March, 1882) has related the case of an inhabitant of Loirett, who having once had a very light attack of splenic fever, had later a second attack of greater gravity, from which he recovered. This writer attributes this result to a sort of preventive vaccination effected by the first malignant pustule. This is but a repetition of what takes place in the case of small pox; a first attack preserves from a second or attenuates it considerably. We might compare these cases with the phenomenon of race habituation and of acclimatization. It is known that the negroes resist yellow fever better than whites. It is known also that the Indo-European does not thrive in Egypt. The races which resist certain contagious diseases, may be considered as naturally vaccinated by their living habitually amid the foci of infection. Acclimatization doubtless has a similar effect.

On their environment depends, then, the life of the bacteria, as does the life of all living beings whatever; an adaptation to this environment is necessary in order that they may thrive and increase or become sufficiently numerous to overcome the resistance of the organic elements. In this adaptation, in this struggle for existence, these micro-organisms undergo modification, attenuation in virulence, and may even become inoffensive, while conferring complete immunity from the deadly diseases which they cause. The temperature of birds opposes an effectual resistance to the multiplication of the anthrax bacillus; the humors of the sheep of Algiers antagonize the existence of the same bacteria, save when introduced in large quantities, while the native sheep are invariably killed by an infection much less in quantity; the asses of Africa also resist the anthracoid virus when inoculated under their skin. In these media, which are not favorable for them, these infinitely little organisms, so powerful and so

terrible in a favorable medium, are vanquished in the struggle for existence. The grand law of the doctrine of evolution here also finds verification, as it does—we may here parenthetically remark—all through nature, as it does in the social life of human races.

It is easy to foresee what progress may be realized in therapeutics if some day a similar process of prevention shall be utilized against the communicable diseases of man.

Here a question presents itself which demands a moment's attention. Can the consumption of anthracoid meat give anthrax? Wagner and Bull of Munich having observed what seemed to be a case of spontaneous anthrax in man (*mycosis intestinalis*), thought that it might have been due to spores or bacilli introduced with the food. But Colin of Alfort pretends that the bacteria are destroyed by the gastric juice as soon as they reach the stomach. What is certain is that Decroix, veterinary surgeon of the army, to triumphantly demonstrate his belief that anthracoid meat does not give splenic fever, did not hesitate to eat it, and ate it with impunity.

But how does it happen that the virus charged with attenuated microbes is no longer infecting? The morbific agent, in these cases enfeebled, has not force enough to struggle successfully against the organic media which oppose it, and ceases to vegetate.

But how do these attenuated viruses give immunity against the invasion of the diseases of which they are the agents? What sort of modifications do they effect in the fluids of an organism naturally congenial to them, so that the non-attenuated microbe can no longer find there a suitable soil for its growth and multiplication? But this is not all: the fœtus in utero is protected from the infectious disease by means of the preventive inoculation of which the mother is the subject, for the fœtus by virtue of its placental connection is an integral part of the mother. Cases of variola communicated by the mother to the unborn child are sufficiently common. So also by the law of heredity the contagious disease may be communicated by the father. Here is one of the chief difficulties against which the microbiotic theory has to contend, viz., the impartation of the animated contagium from the father to the child through the medium of the sperm. Many of these problems in the present state of science are insoluble.

The germ theory, moreover, has contributed to acquaint us with new hygienic data which authorize us to hope the eventual suppression of contagious diseases. What must be done, for instance, to stamp out anthrax? Cremate the bodies of animals that have died of the disease, and destroy or disinfect everything which they have polluted. The old germs, wherever existing, while preserving their virulence for a few years, will eventually disappear altogether.

Recently Fauvel, in the 'Tribune Médicale,' has called attention to the fact that bacteria and mycelia may be found about most nursing bottles from which hand-fed babies are fed. These organisms determine by fermentation the acidity of the milk, and thus have much to do with provoking the intestinal disorders from which so many infants perish. The proper treatment is here clearly indicated: prevent these organisms from developing about nursing bottles; discard the nursing bottle altogether, which is nothing but a nuisance. Authorities, such as Murchison, Buchanan, Radcliffe, etc., have signalized epidemics of typhoid fever, of scarlet fever, of diphtheria, which they have attributed to the use of milk *fouled*

by germs, this milk being brought from farms where the epidemic was prevailing, and simultaneously affecting a great number of persons who made use of it, thus transporting the disease from the infected territory to the city. Pettenkofer has accused potable water of being the bearer of the typhoid contagium. [Is not the fact fresh in our memories of the terrible epidemic of typhoid fever which this very year 1885 raged in Plymouth, Penn., where a mountain stream which fed the reservoir from which the city was supplied with water, was polluted by the dejections of a typhoid fever patient, a great part of a three months accumulation of dejecta being suddenly, during a thaw, swept into the running stream; the consequence was an outbreak of typhoid among citizens making use of this mountain water, resulting in over 1,200 cases of sickness and more than 100 deaths.—Tr.]

A practical lesson from facts on record, proving the transmission of contagious diseases by drinking water and even by milk (here Peuch's cases of the communication of tuberculosis from the cow to man through the milk are in point), is to boil the water or other suspected liquid before using it. It is true, however, that some disease germs resist long boiling.

If the virulent and zymotic diseases are caused by microscopic organisms, it is a laudable endeavor to try to destroy the latter in the organism if they cannot be reached before their introduction. Among the agents given with antiseptic intent, Hamlet (Rev. Sc., No. 11, Sept. 10th, 1881) has found hydrogen, oxygen, nitrogen, carbonic acid, marsh gas, sulphuretted hydrogen without action on the development of microbes. The substances which have arrested the life of these little organisms are: oxygenated water, carbon disulphide, binoxide of nitrogen and chlorine. These conclusions harmonize with those of Pasteur, who lessened the virulent property of the microbes by the action of the oxygen of the air, and with those of Chappuis, who destroyed the germs by ozone.

Potassa, soda, ammonia, bisulphide of potassium, oxalic and benzoic acids, iodide and bromide of potassium, hyposulphite of sodium, tannin and methyl alcohol (5 per cent.) are almost without effect.

The alums, ferrous sulphate, the chlorides of magnesium, iron and aluminum, camphor, salicylic acid, chloroform and phenol (3 per cent.) arrest the multiplication of microbes, but do not destroy them completely. According to Laborde and Bochefontaine, salicylic acid in the proportion of one part to a thousand, prevents all development of the bacteria; mixed with food and drink it is an excellent antagonist of fermentation, and inoffensive even when given in a stronger solution.

Bacchi (Acad. des Sciences, 1879) takes a couple of healthy frogs; he injects under their skin a little blood from the heart of a frog dead from septicæmia. One or two days after, it is remarked that these frogs are very feeble; there is cutaneous hyperæsthesia, their blood globules begin to be deformed, and in their blood is found a considerable number of bacteria animated with active movements. At this moment, under the skin of one of them is injected a small quantity of a solution of phenate of soda; soon afterwards it is observed that in this frog the bacteria, before wriggling in a lively manner, have become motionless, then they disappear altogether; at the same time the globules regain little by little their normal form and at the end of several days the animal recovers its usual health. The other frog, which was not treated by the phenate of sodium, soon dies, presenting all the symptoms of septicæmia.

Dr. White, who believes that yellow fever is the result of a microbe,

has related an interesting incident relative to a means of preservation, put in practice by him. An English ship crew was enabled to sojourn for seven weeks in harbor at Rio Janeiro in a time of epidemic, and right in the face of a hospital full of patients, without a single case of the disease occurring among the sailors, owing to the protection afforded by daily doses of from five to ten grains of salicylic acid; while in 150 schooners anchored in the same harbor, by the side of this vessel, there were in each from two to four deaths.

From all time, it has been a custom to smoke meat to preserve it from putrefaction, and the well-known process of embalming with aromatics has kept corpses from decomposition for thousands of years. In fact the aromatic compounds possess antiseptic properties, and therapeutics has utilized the same agents to oppose the morbid fermentations taking place in the body.

Bichloride of mercury is undoubtedly the most powerful antagonist of putrefaction; then come phenol, quinine, arsenious acid, sulphate of iron, chloride of sodium. The substances which have manifested the most energetic disinfecting properties with regard to choleraic stools are, according to Illisch, nitric acid and phenol. The effects of the following substances have been more feeble: sulphuric and hydrochloric acids, essence of turpentine, wood vinegar, sulphates of copper, zinc, iron, alum, tannin, solution of perchloride of iron, chloride of sodium. According to Pleck, sulphate of alum opposes with the most energy the putrefaction of urine; then come tannin, benzoic acid, salicylic acid, phenol. Bucholtz has observed the resistance of inferior organisms of the same kind (micrococcus, microbacterium, Billroth) in an alimentary liquid always the same (sugar candy, 10 grammes, tartrate of ammonium, 1 gramme, phosphate of potassium 0.05 gramme, in water 100 grammes) and these are the results which he has attained:

Prevent the development of Bacteria.	In the following degree of dilution.	Destroy the Power of Reproduction of Bacteria.	In the following degree of dilution.
Bichloride of Mercury	1 : 20000	Chlorine	1 : 25000
Thymol.....	1 : 2000	Iodine	1 : 5000
Benzoate ol Sodium	1 : 2000	Bromine	1 : 3333
Creasote	1 : 1000	Acid, Sulphurous	1 : 666
Essence of Thyme		" Salicylic	1 : 312
Oil of Caraway....		" Benzoic	1 : 250
Benzoic Acid.....		Thymol.....	1 : 200
Salicylic Acid	1 : 666	Carvol.....	1 : 200
Eucalyptol.....		Sulphuric Acid.....	1 : 161
Essence of Caraway.....	1 : 500	Creasote	1 : 100
Salicylate of Soda..... ..	1 : 250	Phenol	1 : 25
Phenol	1 : 200	Alcohol.....	1 : 4.5
Quinine.....	1 : 200		
Sulphuric Acid.....	1 : 151		
Boracic Acid	1 : 133		
Sulphate of Copper.....	1 : 133		
Hydrochloric Acid	1 : 75		
Sulphate of Zinc	1 : 50		
Alcohol.....	1 : 50		

These results are to be accepted with some reservation, they prove nevertheless the antiseptic power of the aromatic compounds. Many of these substances, with all their capability of opposing morbid fermenta-

tions, are not susceptible of absorption in sufficient doses to act efficaciously, and at the same time with safety to the organic functions.

This is about all that we know respecting the means of prevention, or of treating antiseptically the zymotic diseases.

To sum up, How do the germs of diseases penetrate our systems? By the air which we breathe, and the food which we ingest. The air contains but few virulent germs. In observing the march of epidemics, we note that almost always the persons affected have had more or less direct contact with the sick; epidemics do not seem to travel long distances, as would be the case were the germs wafted hither and thither in the air. It is, however, more likely that the contagious diseases are produced by germs absorbed with drinks and aliments, and as the rule is that heat kills all germs, it is a good plan in times of epidemics to thoroughly cook all food and boil all water before making use thereof. Moreover, when we reflect that a slight modification of the environment prevents bacteria from thriving and multiplying, the possibility is apparent, by a well directed hygiene and appropriate medicaments, to effect such modifications in the organism that the microbes can no longer find a habitat or multiply there. But this is a side of the question hardly yet touched upon.

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